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A NEW ANSWER TO THE QUESTION OF MACULAR SPARING

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According to ordinary field tests, the central visual field is usually spared to a considerable extent when one occipital lobe has been removed or its functions completely destroyed, while the central field is characteristically split vertically when one optic tract is destroyed. If there is actual sparing of the macula in the former case, there seems to be no escape from the conclusion that bilateral representation of the macula exists in the occipital lobes. There is, however, no tenable anatomic evidence in favor of this and in fact much against it.[1] Assumption that the supposed sparing is due to instability of fixation fails to solve the enigma because it does not explain why such sparing is not found when one optic tract is destroyed. The theory that I have to offer explains macular sparing after complete destruction of one occipital lobe as only apparent and as due partly to instability of fixation and partly to the establishment of an eccentric fixation point. In addition it accounts for the instability of fixation after lobar destruction and for absence of apparent sparing after interruption of one optic tract. The theory in brief is that apparent sparing results from loss of necessary cortical integration and that this loss may be produced by cortical injury but not by unilateral interruption of the lower visual pathways. The theory rejects bilateral occipital representation of the macula and is based on the following considerations:

Normally during central fixation of a point in space the image of the point is placed nearly on the center of the fovea. This is known from the fact that visual acuity is highest at the fixation point and falls off symmetrically around it. It has been thought that this placement of the image results from an attempt to see the point most distinctly. This, however, is not the case. For it is a well known fact that when foveal vision has become defective in one eye only, or even completely absent, as in the case of a hole in the fovea, the subject will continue to place on the center of the defective fovea the image of any letter he tries to see with this eye alone. In other words, the subject is unable to see any letter he is "looking at" but can see letters near it. It is evident that, at least in the completed organism, central fixation is not controlled by the high visual acuity of the fovea. It therefore must be controlled by integration and correlation of a wide central region with the ocular motor mechanism. Possibly some of the integration takes place in the retina itself, but obviously some of it, the final part, must take place in the cerebral cortex, and an essential part of the corresponding ocular motor coordination must take place here.

If one looks at a circle on a uniform white background and the circle corresponds in size to the macula, one can accurately fixate the center whether or not an actual

1. Hines, M.: Recent Contributions to Localization of Vision in the Central Nervous System, Arch. Ophth. **28**:913 (Nov.) 1942.

point is there. If half the circle is hidden by a large piece of black paper with a straight edge that passes vertically over the center of the circle, one can still precisely fixate the center with the fovea. Evidently, therefore, if the cerebral cortex is intact, the integration and correlation in question can take place when there is no retinal image on one half of the macula. However, since one completes the circle by the imagination, it can be said that there is an imaginary image on one half of the macula. Obviously, when one optic tract is interrupted an analogous condition exists—one half of the visual field is blocked off while the cerebral cortex remains intact. Hence fixation is normal and splitting of the macular field can be found. This would be the case also if corresponding halves of the retinas were destroyed. When, however, one occipital lobe has been destroyed or removed, and the integration in question is thereby abolished, fixation becomes eccentric and apparent sparing is found. It would seem that the new retinal point of fixation would be at about the center of the remaining macula. This would account for apparent sparing of about three fourths of the whole macula. The remainder of the apparent sparing could be accounted for by fixation slippage dependent largely on the altered correlation with the ocular motor mechanism.

Cases have been reported in which a lesion of one occipital lobe has produced hemianopsia with splitting of the macula. In 1 such case the corpus callosum was cut. In Dandy's 3 cases,[2] in each of which he removed the entire right cerebral hemisphere, it was stated that there was complete hemianopsia.[3] While such cases contradict bilateral representation of the macula in the occipital lobes, they may seem also to contradict my theory as thus far presented. To explain these cases the following elaboration of the theory is offered:

Both halves of the visual field of each eye must be represented somewhere or somehow in one or in each of the cerebral hemispheres. For without some sort of visual communication between the two hemispheres, an image presented only in one hemisphere and another image presented only in the other hemisphere could not be compared. Moreover, the communication must be immediate, because the two images can be immediately compared. Simple as are these facts, and obvious as is their implication, they seem to have been generally disregarded. The phi phenomenon is the same whether the two objects are in the same or in contralateral fields. In some way the two half-fields must be represented as a whole without demarcation between them, for consciously they so appear. Since the two cerebral hemispheres are closely symmetric, it is more than reasonable to believe that a complete visual field is represented in each hemisphere. The site of each representative, which for convenience I shall term the conscious visual area, has not been identified. Possibly it is not in a closely circumscribed area.[4] It is at this level that the theory of replacement[5] assumes that each pair of corresponding retinal units is represented by a single unit. Probably, just as the

2. Dandy, W. E.: Physiological Studies Following Extirpation of the Right Cerebral Hemisphere in Man, Bull. Johns Hopkins Hosp. **53**:31, 1933.

3. It is to be noted, however, that charts of the visual fields are not reproduced and that it is not stated that careful field studies were made. The statement "hemianopsia is of course complete" suggests that completeness was taken for granted and that there may have been apparent sparing in these cases. This question was not discussed by Dandy.

4. The 6 cases reported by A. J. Akelaitis (Studies on the Corpus Callosum, Arch. Neurol. & Psychiat. **45**:788 [May] 1941) in which the corpus callosum was completely sectioned seem to prove that the visual communication between the two hemispheres is not via the corpus callosum, but via the anterior commissure, and therefore suggest that the conscious visual areas are in the temporal lobes.

5. Verhoeff, F. H.: A New Theory of Binocular Vision, Arch. Ophth. **13**:151 (Feb.) 1935.

speech center is situated unilaterally, so the consicous visual area normally employed may be represented unilaterally. In this connection it is of considerable importance that the visual phenomena of squinters who are manifesting anomalous projection can be best explained on the assumption that they are employing one conscious visual area for the right eye and the other for the left eye. Since fixation is largely a voluntary act, it is reasonable to assume that the integration necessary for this act and for conscious visual acuity largely takes place in a conscious visual area, and that in the case of right-handed persons this is usually in the left cerebral hemisphere.

Integration between the two fields would be possible so long as the conscious visual area in use and its motor connections remained intact. As has been pointed out, the integration in question can take place when in one field there is no image from an external source. Therefore splitting of the macular field could be found even after removal of one occipital lobe and complete severance of all connections between the two cerebral hemispheres. If, however, in the conscious visual area used, the function of one macular half was destroyed or seriously impaired, apparent sparing of the macula could be found as I have explained, whether or not the lower visual pathways to this half were intact. Obviously, if the conscious visual area concerned was in the left hemisphere, removal of the left occipital lobe would be more likely to injure, directly or indirectly, the left half of this area than would removal of the right lobe. It is noteworthy, therefore, that of the 2 cases reported by Halstead and associates [6] alleged sparing occurred only in the 1 in which the left lobe was removed. In the case in which the right lobe was removed and no sparing was found, it was definitely stated that the patient was right handed, but in the other case no statement was made as to this question. It may be of importance, also, that the case in which there was no apparent sparing was the one in which it was not certain that all of the striate area had been removed. In a case of hemianopsia, my theory predicts that the less the occipital damage the less the probability of apparent macular sparing. Of course, for real sparing the opposite is true.

Actual sparing without bilateral occipital representation of the macula could occur after complete destruction of one occipital lobe only if visual pathways to the conscious visual area in use could by-pass the injured occipital region. For certain lower animals this may be possible, but for man it would seem not to be so. Yet a case of a boy aged 11 years has been reported [7] in which one occipital lobe was found at operation to have been completely replaced by a cystic cavity without impairment of visual acuity and without production of hemianopsia or even of important constriction of the visual field. It is to be noted, however, that in this case the lesion began early in life, as the result of birth injury, and that therefore the case is not comparable to cases in which the lesion was acquired much later. Moreover, although discussed, the possibility was not completely excluded that the occipital cortex had simply been pushed aside by the cyst.

Hence if actual sparing of the macula occurred in the case of total lobectomy reported by Halstead and associates,[6] my explanation is nullified. They took unusual precautions to avoid error. By an electrical method they excluded fixation movements of more than 30 minutes of arc. They also employed a photographic method, but did not state how accurate this was. Presumably it was less

6. Halstead, W. C.; Walker, A. E., and Bucy, P. C.: Sparing and Non-Sparing of Macular Vision Associated with Occipital Lobectomy in Man, Arch. Ophth. **24**:948 (Nov.) 1940.

7. Jelsma, F.; Spurling, R. G., and Freeman, E.: Absence of Occipital Lobe of Brain (Porencephaly) with Essentially Normal Vision, Arch. Neurol. & Psychiat. **28**:160 (July) 1932.

so than the electrical method, for they did not emphasize it. While it seems certain that they excluded the possibility of ordinary fixation movements during the field tests, they presented no evidence to exclude the possibility that a gradual slipping of fixation occurred when the patient anticipated that a test object was to be brought from the blind half of the macular field. As a matter of fact, in his original description of his electrical method, Halstead [8] made the following statement: "The amplitude of eye movements of slower rate than the time constant of the amplifier (approximately one second) will of course be incompletely recorded." It is just such slippage that my theory predicts, and little of it would be required to explain their case on this basis alone. These investigators by refined methods demonstrated sparing of only 1.25 degrees for form and 2.5 degrees for brightness. Other observers by the usual methods repeatedly found sparing of 3 to 5 degrees in the same case. This fact of itself suggests that the extent of apparent sparing varied inversely with refinement of technic and that therefore no actual sparing existed. It is to be noted that in this case the visual acuity, normal in each eye before operation, was reduced in the right eye to 0.8 and in the left eye to $0.8 + 2$. This would not be expected if there was actual sparing of the foveas. Apparently also of significance is the fact that in their second case these investigators by means of their refined technic demonstrated splitting, while by usual methods they sometimes found sparing of as much as 2 degrees. This fact accords with the view that there was no actual sparing in either case.

A fixation point eccentric to the extent of 4 degrees of arc would of course be associated with poor visual acuity. Probably an eccentricity of 1.5 degrees in an otherwise normal eye would reduce visual acuity to at least 20/30. How, then, can the fact be explained that in many of the cases under consideration central visual acuity was slightly if at all affected? It is true, as I have indicated, that a person who has acquired defective foveal vision in one eye only will fixate with the defective fovea when the other eye is occluded. In such a case, however, there is ordinarily no reason for him to learn to do otherwise, since the other eye is not defective, and in binocular vision retinal correspondence causes normal bifixation. The circumstances are different when there is hemianopsia with eccentric fixation. Then no doubt the patient soon discovers, consciously or subconsciously, that he must look to one side of an object to see it distinctly and therefore does so when his visual acuity is tested and the best is insisted on. On the other hand, in a visual field test it is not the fixation object but the approaching test object that he is desirous of seeing distinctly. Hence he permits the image of the fixation object to occupy the eccentric macular area or to slip still farther from the real fovea.

There remains to be explained why apparent sparing does not extend above and below the macula. This can be explained on one or all of three reasonable assumptions. The first is that fixation would become less eccentric, that is, approach the normal, when the necessary attention was given to a test object above or below the fixation point. The second is that the subject would consciously or subconsciously discover that he could not make the approaching test object appreciably more distinct by permitting the fixation object to become less distinct. The third assumption is that at a distance from the fovea loss of integration across the margin of the blind field would produce a wider zone of impaired visual perception than it would near the fovea. Thus when a small test object

8. Halstead, W. C.: A Method for the Quantitative Recording of Eye Movements, J. Psychol. **6**:177, 1938.

was brought in from the blind field along the horizontal midline, it would be seen sooner than when it was brought in from above or below the fixation point. To elucidate this question further, and to evaluate the relative importance of these assumptions, field tests other than those which have already been used in such cases would need to be employed.

W. Fuchs [9] has described what he terms a pseudofovea as occurring in certain cases of hemianopsia. By this he meant a retinal area which has a higher visual acuity than the real fovea. While he intimated that he has made similar observations in other cases, he rested his contention on 1 case, to which he devoted a separate lengthy report.[9b] He stated that there was no sparing of the macula in this case, but here he seems to have begged the question. For all the results he obtained can best be explained on the assumption that there was actual sparing. Moreover, it is impossible to determine the extent of the damage to the occipital lobe in this case or in any of the 3 previous cases he reported.[9a] He described no case in which one occipital lobe had been completely destroyed and did not discuss the question as to whether the site of the lesion concerns the pseudofovea. For these reasons, his findings, while interesting from a psychologic viewpoint, cannot be regarded as of important significance in connection with the subject of bilateral representation of the macula. The most that can safely be said of them in this connection is that they do not controvert my explanation of apparent sparing, and possibly lend some support to it.

CONCLUSIONS

Apparent sparing of the macula after complete destruction of one occipital lobe is explained by loss of integration between the seeing field and the blind field in the conscious visual cortical area in use. As a result of this loss an eccentric retinal point is employed for fixation, and slipping of fixation easily occurs. The original macular field is actually split through the center but in field tests appears to be more or less spared.

When one optic tract is interrupted, the integration in question is not disturbed, and splitting of the macula is therefore found in field tests. Even after complete destruction or removal of one occipital lobe, macular splitting may be found in such tests if integration in the conscious visual area in use is not too greatly disturbed. These conditions would most likely obtain when the lobe destroyed was on the side opposite that of the conscious visual area.

This theory needs additional facts before it can be unreservedly accepted. Nevertheless, it seems sufficiently satisfactory to warrant the statement that the field determinations thus far made do not refute the evidence against bilateral occipital representation of the macula.

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9. Fuchs, W.: (*a*) Untersuchungen über das Sehen des Hemianopiker und Hemiamblyopiker, Ztschr. f. Psychol. **84**:67, 1920; (*b*) Eine Pseudofovea bei Hemianopikern, Psychol. Forsch. **1**:157, 1921.

PENICILLIN AND SULFADIAZINE

IN THE TREATMENT OF EXPERIMENTAL INTRAOCULAR INFECTION WITH PNEUMOCOCCUS

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Diplococcus pneumoniae is considered the most frequent cause of severe intraocular infections following perforating injuries. Morax and Moreau,[1] A. von Szily [2] and K. Lindner [3] stressed the importance of this organism in infections of penetrating wounds of the eye in modern warfare. Therefore the chemotherapy of experimental post-traumatic endophthalmitis caused by the pneumococcus was selected for the present study, which is the first part of more extensive investigations on the treatment of intraocular infections.

Statistical data on the types of pneumococci in cases of endophthalmitis and panophthalmitis following various perforating injuries are not available. Only in a few such cases have the types been identified according to Cooper's classification. It is justifiable, however, to apply to intraocular infections the statistical figures of the incidence of the various pneumococcic types present on the normal conjunctiva and isolated in external diseases of the eye, because it is assumed that the micro-organisms from the surface of the conjunctiva and cornea, drawn into the eye by the penetrating foreign body, generally become the source of infection. There was almost complete agreement among the investigators of different countries until 1938 that pneumococci in group IV of the former nomenclature were the most often found and were followed in incidence by strains of type III (Cheney, 1922 [4]; Lundsgaard, 1925 [5]; Vita, 1926 [6]; Wright, 1927 [7]; Clegg, 1927 [8]; Lucic, 1927 [9]; Lobeck, 1931 [10]; Jahnke and Wamoscher, 1931 [11]; Schmelzer and Eckstein,

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1. Morax, V., and Moreau, F.: Étiologie des blessures oculaires par projectiles de guerre, Ann. d'ocul. **153**:321, 1916.
2. von Szily, A.: Atlas der Kriegsaugenheilkunde, Stuttgart, F. Enke, 1916.
3. Lindner, K.: Ueber die Infektionsgefahr nach bulbuseröffnenden Operationen, Ztschr. f. Augenh. **53**:305, 1924.
4. Cheney, R. C.: Types of Pneumococcus Found in Corneal Ulcers, Tr. Internat. Cong. Ophth. **1**:378, 1922.
5. Lundsgaard, K. K. K.: Pneumococcus Infections of Eyes, Hospitalstid. **68**:505, 529 and 553, 1925.
6. Vita, A.: La classificazione immunologica moderna dei pneumococchi nella patologia oculare, Società italiana di oftalmologia, 1926; abstracted, Zentralbl. f. d. ges. Ophth. **19**:712, 1927-1928.
7. Wright, R. E., in Etiology and Treatment of Hypopyon Ulcer Symposium, Tr. Ophth. Soc. U. Kingdom **47**:72, 1927.
8. Clegg, J. G., in Etiology and Treatment of Hypopyon Ulcer Symposium, Tr. Ophth. Soc. U. Kingdom **47**:72, 1927.
9. Lucic, H.: Bacteriology of the Normal Conjunctival Sac, Am. J. Ophth. **10**:829, 1927.
10. Lobeck, E.: Zur Frage der Pneumokokken Infektionen am Auge, Arch. f. Ophth. **127**:395, 1931.
11. Jahnke, W., and Wamoscher, L.: Zur Frage der Serumtherapie bei Pneumokokkenerkrankungen des Auges, Ztschr. f. Augenh. **74**:215, 1931.

1934[12]; McKee, 1935,[13] and Spiratos, 1937[14]). In ophthalmology the first attempts to identify the twenty-nine higher types of the former group IV by agglutination reaction were carried out by Newman,[15] Woods[16] and Chamberlain.[17] These authors arrived, however, at slightly different conclusions. Newman listed as most often found types VII, X and XXIII; Woods, types VII, XIV and XXIII, and Chamberlain, types XIX, VI and XIV.

Since in a few clinical cases of panophthalmitis types III, VII and X were reported as the cause of the infection and since these types are among those most frequently found on the surface of the eyeball, they were selected for inoculation into rabbits' eyes. Furthermore, the effect of one of the drugs studied in experiments on rabbits was tested in vitro on the other frequently found types—namely, types VI, XIV, XIX and XXIII.

By injections of a dilution of a twenty-four hour broth culture into the anterior chamber of adult chinchilla rabbits a fairly well standardized infection with a typical course was obtained and used as the test object.[18] The action of two chemotherapeutic agents was studied, sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine), as one of the newer sulfonamide compounds, and penicillin, a product of the mold Penicillium notatum.

PRELIMINARY EXPERIMENTS

The result of injection of different micro-organisms into the anterior chamber has been studied by several authors, notably by Picot,[19] in 1898, who gave a good description of the clinical and anatomic changes, and by Venco[20] in his study on the effect of sulfonamide compounds on streptococcic and pneumococcic infections. The technics applied by these investigators were different from each other and also from that reported here. In Picot's experiments no attempt was made to estimate the number of germs injected. Venco used dilutions of a dried powder of pneumococci with an unknown number of viable germs. In the experiments reported here this number was roughly estimated in each series by plate counts of appropriate dilutions of the broth culture in blood agar. In the first part of the preliminary experiments the course of the inflammation after injection of the various types of pneumococci was observed. The inflammatory signs, including excessive fibrinous or cellular exudation, usually reached their height in forty-eight to seventy-two hours. With the strain of type VII a violent endophthalmitis with a massive

12. Schmelzer, H., and Eckstein, E.: Die augenpathogene Bedeutung der Streptokokken und Pneumokokken (547 Untersuchungen), Arch. f. Ophth. **132**:24, 1934.

13. McKee, S. H.: A Study of the Pneumococcus Group from the Inflamed Conjunctiva and Lacrimal Sac, Am. J. Ophth. **18**:1021, 1935.

14. Spiratos, S.: Le pneumocoque, agent pathogène et saprophyte de l'oeil, Arch. d'opht. **1**:589, 1937.

15. Newman, E. W.: Diplococcus Pneumoniae and Streptococcus Viridans in Ocular Diseases, Arch. Ophth. **19**:95 (Jan.) 1938.

16. Woods, M. A.: A Study of the Methemoglobin-Producing Organisms in Ocular Inflammations, Am. J. Ophth. **22**:1111, 1939.

17. Chamberlain, W. P., Jr.: The Treatment of Pneumococcic Corneal Lesions with Sulfapyridine, Thesis, Columbia University Graduate School, 1940.

18. Vaccination of the cornea with intracorneal injections was not satisfactory with our technic and culture material because of the frequent spontaneous regressions.

19. Picot, V.: Recherches expérimentales sur l'inoculation de micro-organismes dans la chambre antérieur de l'oeil du lapin, Arch. d'opht. **18**:341, 1898.

20. Venco, L.: Ricerche sulla attività chemioterapica della para-aminofenilsulfamide nelle infezioni microbiche sperimentali dell'occhio, Ann. di ottal. e clin. ocul. **67**:179, 1939; Rilievi sperimentali sulla chemioterapia delle infezioni pneumococciche oculari mediante alcuni derivati organici dello zolfo, Arch. di ottal. **46**:307, 1939.

exudate was observed within twelve hours. The character of the exudate and the sequence of the clinical changes varied somewhat with the strain of the pneumococcic type, but the inflammation generally led to suppuration of the anterior segment, perforation and phthisis bulbi or severe endophthalmitis followed by atrophy of the eyeball. Often in the later stages the cornea became partially or entirely infiltrated and vascularized. Of the 31 control eyes 1 healed spontaneously after a regression of iritis with hypopyon, and in another eye the infection did not take. The stages of the infection were routinely studied with the corneal microscope and slit lamp as long as massive exudate did not fill the anterior chamber. A large number of photographs were taken to illustrate the course of the infection.

On the basis of previous experiments on Bacillus pyocyaneus keratitis [21] and clinical experience, sodium sulfadiazine was used iontophoretically in a 5 per cent solution. The amount of 0.25 Gm. per kilogram of body weight of sulfadiazine fed once daily was chosen to approximate the dose in human therapy. The second part of the preliminary experiments was concerned with the determination of the concentrations of the ammonium or sodium salt of penicillin [22] best suited for local use. Solutions of 0.1 to 1.0 per cent were introduced by corneal baths and by iontophoresis.[23] With both technics solutions of 1.0 and 0.5 per cent damaged the epithelium and superficial layers of the cornea. Solutions of 0.25 per cent also damaged the epithelium to some extent when applied repeatedly by iontophoresis at 2 milliamperes for five minutes. The same concentration usually did not harm the cornea when applied frequently in a simple bath. A solution of 0.1 per cent could be employed with iontophoresis repeatedly with little or no damage to the epithelium. The two latter concentrations were therefore chosen for the experiments. The hydrogen ion concentration of these solutions was found to be between 7 and 8. The injurious action of the higher concentrations on the corneal epithelium has no parallel in the experiments on tissue cultures and on leukocytes reported by Florey and associates,[24] Thygeson [25] and others.

EXPERIMENTAL TECHNIC

The experiments were divided into two groups. In the first group a sharp 27 gage needle was inserted through the periphery of the cornea in a tangential direction, and 0.05 cc. of diluted broth culture of types III, VII or X was introduced into the anterior chamber. With this technic, visible loss of fluid rarely occurred after the withdrawal of the needle. In the second group the pupil was dilated with atropine sulfate solution, the anterior lens capsule injured with the needle and the aqueous permitted to escape. The needle was left in situ, and 0.07 cc. of the inoculum broth was then injected. Actually 0.05 cc. was introduced, since the measured capacity of the needle was 0.02 cc. In this group it was usually not possible to prevent the loss of an uncontrolled amount of fluid through the needle track in the cornea after the injection because the movement of the needle in the extensive slitting of the lens

21. von Sallmann, L.: Sulfadiazine Iontophoresis in Pyocyaneus Infection of Rabbit Cornea, Am. J. Ophth. **25**:1292, 1942.

22. Dr. Karl Meyer and his co-workers provided a regular supply of penicillin. It was prepared in his laboratory from the culture of Penicillium notatum by chloroform extraction and precipitation of the free acid by light purified petroleum benzine (Meyer, K.; Chaffee, E.; Hobby, G. L.; Dawson, M. H.; Schwenk, E., and Fleischer, G.: On Penicillin, Science **96**:20, 1942). In most instances a weighed amount of the free acid was converted to the sodium salt by adding a drop of alcohol and finally dissolving the material in sodium bicarbonate-carbon dioxide buffer.

23. The technic of the corneal bath and that of iontophoresis were the same as those used in experiments on B. pyocyaneus keratitis.[21]

24. Florey, H. W.; Abraham, E. P.; Chain, E.; Fletcher, C. M.; Gardner, A. D.; Heatley, N. G., and Jennings, M. A.: Further Observations on Penicillin, Lancet **2**:177, 1941.

25. Thygeson, P.: Personal communication, cited by Hobby, G.; Meyer, K., and Chaffee, E.: Chemotherapeutic Activity of Penicillin, Proc. Soc. Exper. Biol. & Med. **250**:285, 1942.

capsule enlarged the opening. All experiments and treatments were performed with the eye under anesthesia induced by 0.1 per cent nupercaine hydrochloride.

The size of the inoculum as estimated by plate counts of appropriate dilutions of the broth culture in each series varied widely. The minimal number of organisms injected was calculated to be 250 to 70,000. In general, 0.05 cc. of a 10^{-4} dilution of a twenty-four hour broth culture was given. The purity of the culture was repeatedly controlled by plating and the types with specific antiserums. The virulence of type III, which was used over a longer period, was increased several times by passage through mice.

Treatment was started six and twelve hours after inoculation in the first group and six hours after inoculation in the second group. The inflammatory symptoms after six hours in the first group varied, chiefly according to the number of organisms and the virulence of the strain, from the presence of a marked Tyndall sign and numerous circulating and deposited cells to that of fibrinous or cellular exudate which covered parts of the pupil and of the iris. The signs twelve to thirteen hours after injection were generally those of a much more advanced iritis. The inflammation due to type VII was especially violent. In about 60 per cent of the experiments with this type the anterior chamber was almost completely filled with a fibrinous or gray opaque exudate at the time of the first treatment. The severity of the infection was indicated by marked chemosis. In 2 control experiments in which 0.05 cc. of sterile broth was injected into the anterior chamber, a few cells were seen in the aqueous and on the surface of the lens for about twenty-four hours. In the second group a fibrous net-

TABLE 1.—*Results of Sulfadiazine and Penicillin Treatment Begun Six Hours After Injection of D. Pneumoniae Types III and X*

Treatment	Type of D. Pneumoniae Injected	Approximate Number of Organisms Injected	Number of Eyes	Period of Treatment	Number of Recoveries	Number of Temporary Improvements	Number of Losses
Sodium sulfadiazine iontophoresis and sulfadiazine feeding	III	1,000	6	2-4 days	..	..	6
	X	270	3	2-4 days	1	2	..
		2,120	3	2-4 days	..	..	3
Control eyes—influenced only by sulfadiazine given orally	III	1,000	2	2-4 days	..	..	2
	X	270	2	2-4 days	..	..	2
		2,120	2	2-4 days	..	..	2
Sodium penicillin iontophoresis or bath	III	1,000	5	2 days	5	..	..
	X	270	3	2-3 days	3	..	..
		2,120	3	2-3 days	3	..	..
Control eyes—no treatment	III	1,000	3	.....	..	..	3
	X	270	1	.....	..	..	1
		2,120	1	.....	..	..	1

work covered the cataractous lens in 8 uninoculated control eyes as well as in the inoculated eyes. However, in the latter the other signs of inflammation were generally fully developed. In all instances in which the inflammation was unequal, the worse eye was treated and the better retained as a control.

The severity of the clinical picture governed the period of treatment as well as the length and number of the iontophoretic applications and of the corneal baths. In the six hour experiments one treatment of five minutes was given on the first day, repeated on the morning of the second day and supplemented by a three minute application in the afternoon. In the twelve to thirteen hour experiments the eyes were treated twice on the day following the inoculation for five and three minutes, respectively. Treatment was continued from two to six days in all experiments. Generally the number of applications of penicillin and the application time were reduced on the second or third day, and further therapy depended on the response of the infection to the treatment. As a rule equal numbers of eyes were treated in each series with the corneal bath, with ionization in which 0.01 per cent solution was used and with ionization in which 0.25 per cent solution was used. In the second series of the infections with type VII, therapy starting twelve to thirteen hours after inoculation, only iontophoresis was employed.

RESULTS

First Group.—Table 1 shows that local application and oral administration of sulfadiazine started six hours after inoculation generally did not prevent the development of a severe endophthalmitis. The high concentration of the drug in the aqueous attained with iontophoresis controlled the infection in several instances

for two to three days, but was evidently not effective enough after this period to prevent flare-ups which finally led to destruction of the eyeball. There were 3 exceptions. Of twelve eyes, in only 1, which received a small inoculum, did the inflammation clear up completely. In one rabbit the condition of both eyes improved considerably but an insidious iritis persisted. The 6 control eyes, which were influenced only by general treatment, suppurated. The results were the same with type III and type X.

Penicillin applied six hours after inoculation stopped the infection with types III and X in all instances—that is, in 11 eyes. No relapses occurred. The treatment was continued on the average of two to four days. The 5 control eyes were destroyed by the infection.

Table 2 presents the results of a series of experiments with types III, VII and X in which there was a lapse of twelve to thirteen hours between the inoculation and the treatment. No attempt was made to use sulfadiazine because of the unfavorable results at an earlier stage of the inflammation. In 25 of the 30 eyes

TABLE 2.—*Results of Penicillin Treatment Begun Twelve to Thirteen Hours After Injection of D. Pneumoniae (Types III, X and VII)*

	Type of D. Pneumoniae Injected	Approximate Number of Organisms Injected	Number of Eyes	Period of Treatment	Number of Recoveries	Number of Losses
Treated Eyes						
	III	360	6	2-3 days	5	1
		1,360	6	2-3 days	6	..
	X	4,300	6	3 days	5	1
	VII	1,500	6	5 days	5	1
		2,200	6	4 days	4	2
Totals			30	.....	25	5
Control Eyes						
	III	360	1	.....	1	..
		1,360	2	.....	1	1
	X	4,300	2	.....	..	2
	VII	1,500	2	.....	..	2
		2,200	2	.....	..	2
Totals			9	.....	2	7

in the series penicillin therapy was effective. In more than half of the eyes in the most advanced stages of endophthalmitis intensive treatment resulted in a final cure (fig. 1), even when a transient rise in the intraocular pressure had increased the dimensions of the anterior segment of the eyeball. The ectasia bulbi regressed with the improvement of the inflammation, as the photographs in figure 2 show. In the lost eyes the endophthalmitis ran the following course: In an eye infected with type III and treated with a corneal bath the inflammation flared up on the third day and could not be controlled by more vigorous treatment. The second failure occurred in an eye which had received an inoculum of approximately 4,300 organisms of type X; iontophoretic treatment did not prevent the development of an abscess in the vitreous, and the eye was subsequently lost. In the 3 remaining eyes which were lost, the violent inflammation caused by the very virulent strain of type VII was apparently too advanced to be improved by the therapy. Of 9 control eyes, 7 were destroyed. In 1 control eye the infection did not take, and in the other the iritis with hypopyon cleared up spontaneously.

Second Group.—Because of the discouraging results with sulfadiazine in eyes without injury to the lens the experiments with traumatic cataract were confined to the penicillin treatment of eyes infected with types III and X. Of 12 eyes inocu-

lated with type III and treated with penicillin, 3 were lost (table 3). The 3 failures belonged to the first tentative experiments, carried out before it was learned that, even if the inflammation appears checked macroscopically, intensive treatment should be continued for a few days. It is probable that these eyes could have been saved if iontophoretic treatment had been prolonged beyond the third

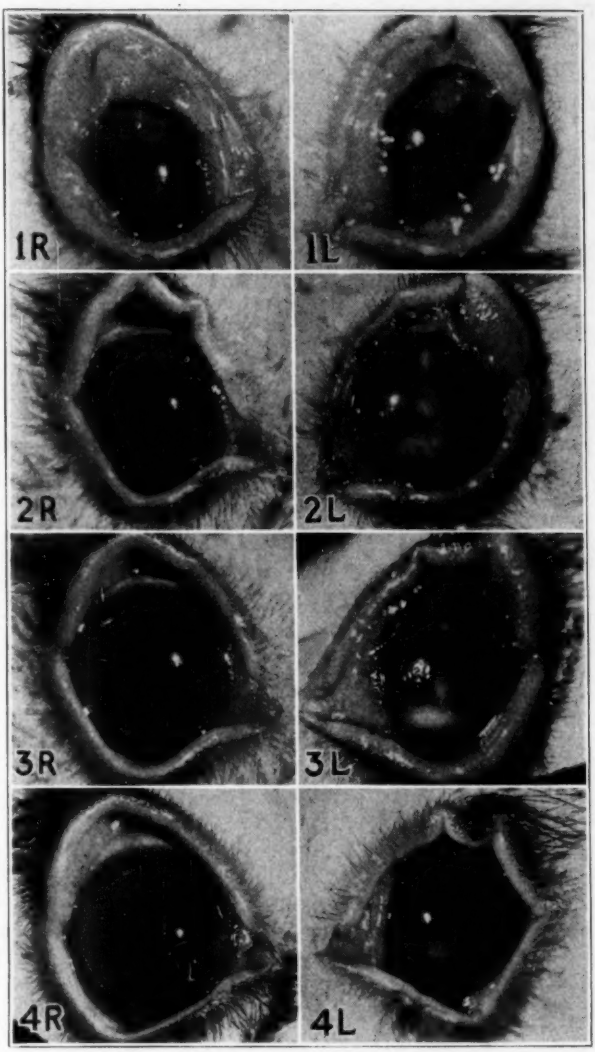

Fig. 1.—Effect of penicillin treatment begun twelve hours after inoculation with D. pneumoniae of type VII. *R* indicates the treated and *L* the untreated eye; 1 *R* and 1 *L* were taken prior to the treatment and 2 *R* and 2 *L*, 3 *R* and 3 *L* and 4 *R* and 4 *L* after one, two and four weeks, respectively.

day. Three control eyes were lost in the usual course of infection. Excellent results were obtained in the 9 eyes inoculated with type X and treated with penicillin, even though they presented advanced stages of infection and in 2 instances a fully developed endophthalmitis at the beginning of the treatment. Two of the

3 control eyes showed a stage of panophthalmitis after twenty-four hours, and all control eyes were destroyed.

Experiments on Other Types.—In order to consider the more frequently found types not studied in experiments on rabbits, types VI, XIV, XIX and XXIII were examined in vitro for their sensitivity to penicillin. Fleming's [26] dilution method was employed in accordance with Hobby's [27] demonstration of its superiority over the Oxford ring technic for sterile material. In all examined types growth was inhibited in the same range with the same sample of penicillin salt. The results did not differ appreciably from those obtained in vitro with types III and VII. Preparations of the penicillin salt routinely showed an activity of 0.15 to 0.3 microgram per cubic centimeter against 2,000,000 to 4,000,000 pneumococci of single strains of the various types.

COMMENT

Fleming, in 1929, first noted the antibacterial action of a strain of a Penicillium notatum against many gram-positive organisms. He called the active agent

TABLE 3.—*Results of Penicillin Treatment Begun Six Hours After Injury of the Lens and Injection of D. Pneumoniae (Types III and X)*

	Type of D. Pneumoniae Injected	Approximate Number of Organisms Injected	Number of Eyes	Period of Treatment	Number of Recoveries	Number of Losses
Treated Eyes						
	III	800	6	4 days	3	3*
		960	6	4-5 days	6	..
	X	42,000	6	5-6 days	6	..
		75,000	3	5-6 days	3	..
Totals			21	.....	18	3
Control Eyes						
	III	800	2	.....	..	2
		960	1	.....	..	1
	X	42,000	2	.....	..	2
		75,000	1	.....	..	1
Totals			6	.....	0	6

* One eye was treated by corneal bath, 1 iontophoretically with 0.1 per cent solution of penicillin salt and 1 iontophoretically with 0.25 per cent solution of penicillin salt.

penicillin and suggested its application to infections due to susceptible organisms. In the last three years the chemotherapeutic activity of penicillin was extensively studied in vitro and in vivo by several groups of investigators (Chain and associates,[28] Florey and collaborators [24] and Dawson and associates [29]). Florey and his co-workers pointed out a great strain difference in the sensitivity of pneumococci to penicillin. Hobby and her collaborators [30] found that pneumococci were destroyed more rapidly than hemolytic streptococci. Among the studies of the clinical use of penicillin is the report of Florey and associates of the effective

26. Fleming, A.: On the Antibacterial Action of Cultures of a Penicillium, with Special Reference to Their Use in the Isolation of B. Influenzae, Brit. J. Exper. Path. **10**:226, 1929.

27. Dr. Gladys Hobby gave practical advice in the application of this method.

28. Chain, E.; Florey, H. W.; Gardner, A. D.; Jennings, M. A.; Orr-Ewing, J., and Sanders, A. G.: Penicillin as a Chemotherapeutic Agent, Lancet **2**:226, 1940.

29. Dawson, M. H.; Hobby, G. L.; Meyer, K., and Chaffee, E.: Penicillin as a Chemotherapeutic Agent, J. Clin. Investigation **20**:434, 1941.

30. Hobby, G. L.; Meyer, K., and Chaffee, E.: Activity of Penicillin in Vitro, Proc. Soc. Exper. Biol. & Med. **50**:277, 1942.

treatment of corneal ulcer in 2 cases and of conjunctivitis in 2 cases. So far as I know, other clinical or experimental observations on penicillin therapy of infections of the bulbus have not been published to date.

The results of the penicillin treatment of experimental intraocular infections with strains of pneumococci of types III, VII and X and the notable antibacterial

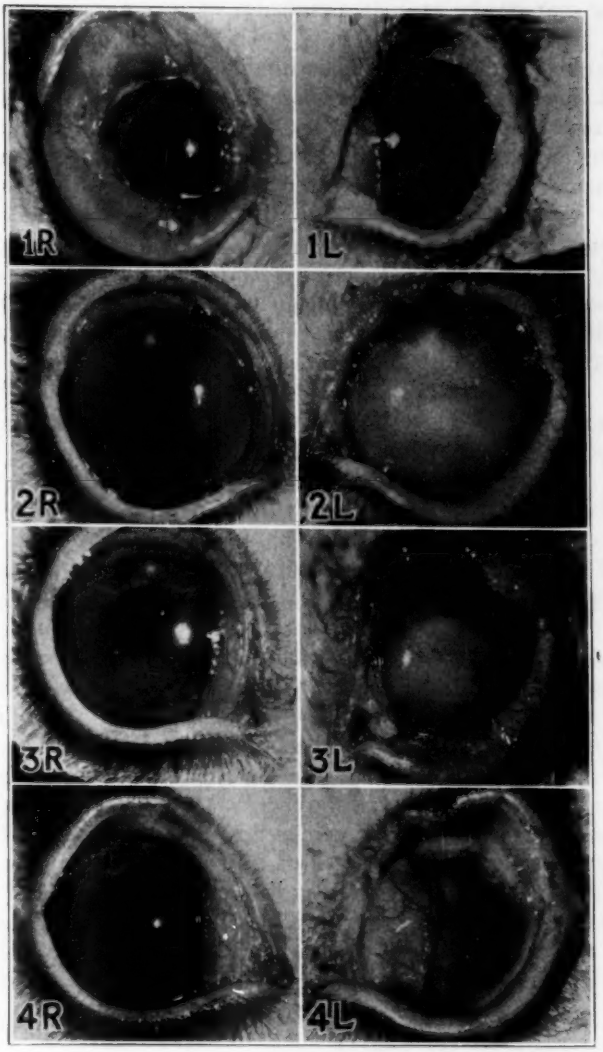

Fig. 2.—Effect of penicillin treatment begun twelve hours after inoculation with D. pneumoniae of type VII. *R* indicates the eye successfully treated despite development of secondary glaucoma with enlarged anterior segment and *L* the untreated eye; 1 *R* and 1 *L* were taken prior to treatment and 2 *R* and 2 *L*, 3 *R* and 3 *L* and 4 *R* and 4 *L* after one, two and four weeks, respectively.

action of penicillin in vitro on strains of the other types frequently found in the eye justify an optimistic point of view in regard to the clinical application of penicillin in the treatment of ocular pneumococcic infection. The activity of penicillin in severe pneumococcic endophthalmitis will be more appreciated when it is

compared with the failures of sulfadiazine treatment in the present series. These have their analogy in the unsatisfactory results of general sulfapyridine (2-[para-aminobenzenesulfonamido]-pyridine) therapy in experimental pneumococcic ulcers in rabbits as reported by Chamberlain.[31] Venco studied sulfonamide compounds in the treatment of experimental intraocular infections with pneumococci of types I, II and III. Treatment was begun at the time of infection, but the results in his small series can scarcely be evaluated, because of the questionable course in the control eyes.

The clinical effect of sulfapyridine treatment in 2 cases of pneumococcic ulcer was called of doubtful significance by Guyton and Woods.[32] In 4 of 6 cases of pneumococcic corneal ulcer observed by Chamberlain [31] systemic treatment with sulfapyridine was apparently without effect, and in the remaining cases the results were not convincing. Bellows [33] mentioned 2 cases of pneumococcic serpent ulcer in which early chemotherapy with sulfonamide compounds did not stop the progression. On the other hand, the first favorable reports by Bailey and Saskin,[34] Roggenkämpfer [35] and Rosengren [36] on the treatment of serpent ulcer with sulfonamide compounds cannot be judged adequately because of the absence of bacteriologic identification. In 1 case of pneumococcic endophthalmitis Guyton [37] found sulfanilamide therapy ineffective.

In some of the present series of experiments the initial good effect of the combined sulfadiazine treatment lasted for a few days. But usually a heavy exudate developed later and no further treatment could prevent the destruction of the eye. In a rabbit infected with type III, the inflammation became worse in spite of intensive sulfadiazine treatment of both eyes and feeding of sulfadiazine over a period of four days. Then penicillin was applied to the more severely affected eye. The iritis with hypopyon and other signs of inflammation cleared up after two treatments, and the eye returned to normal after six days. The other eye was destroyed by the infection. In a second rabbit combined sulfadiazine treatment greatly improved the inflammation in both eyes, but an insidious iritis persisted, with numerous cells in the anterior chamber, swelling and hyperemia of the iris and posterior synechias. Two applications of penicillin to one eye nineteen days after inoculation and to the other thirty-five days after inoculation cleared the anterior chamber of the residual symptoms. Florey and his collaborators demonstrated the significant differences between the action of the sulfonamide compounds and penicillin. Of special bearing is the finding that hydrolytic protein breakdown products or products of tissue autolysis or pus do not affect to any appreciable degree the bacteriostatic action of penicillin, whereas they annul that of the sulfonamide compounds in vitro. Whether this difference, in addition to the higher bacteriostatic power of penicillin, explains the dissimilar effects of these two forms of chemotherapy in the eye cannot be decided.

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31. Chamberlain, W. P., Jr.: Treatment of Pneumococcic Corneal Lesions with Sulfapyridine, Arch. Ophth. **27**:869 (May) 1942.

32. Guyton, J. S., and Woods, A. C.: Advances in the Use of Sulfanilamide Compounds in Ophthalmology, Am. J. Ophth. **24**:428, 1941.

33. Bellows, J. G.: Chemotherapy in Ophthalmology, Tr. Am. Ophth. Soc. **47**:19, 1942.

34. Bailey, J. H., and Saskin, E.: Treatment of Corneal Ulcer with Sulfanilamide, Arch. Ophth. **22**:89 (July) 1939.

35. Roggenkämpfer, W.: Hornhautulkus und Prontosil, Klin. Monatsbl. f. Augenh. **103**: 211, 1939.

36. Rosengren, B.: Treatment of Ulcus Serpens Corneae with M. & B. 693, Acta ophth. **17**:209, 1939.

37. Guyton, J. S.: The Use of Sulfanilamide Compounds in Ophthalmology, Am. J. Ophth. **22**:833, 1939.

Emphasis must be placed on the results of experiments with a twelve to thirteen hour interval between the inoculation and the first treatment with penicillin. At this time, in which the end of the logarithmic multiplication of the organisms would be reached in vitro, the structures of the anterior chamber show marked changes, such as swelling and hyperemia of the iris, massive exudate of plastic and fibrinous type and synechias. Chemosis is often pronounced. In one eye in which perforation occurred after twenty-four hours, continued treatment led to an excellent result.

Half of the few trials of local treatment with penicillin carried out when the clinical picture of suppuration of the anterior segment developed or the anterior chamber was almost completely filled with exudate were without success. Two eyes with abscess of the vitreous also did not respond. In this connection it is of interest that the vitreous fluid of normal eyes after local application of penicillin did not demonstrate any bacteriostatic activity in vitro.

According to common clinical experience and the histologic observations of K. Lindner,[3] the prognosis for post-traumatic intraocular infections is much less favorable when the lens is injured. In the experiments in which the lens was damaged (group II) the course of the inflammation was somewhat more serious than in the experiments with uncomplicated infections (group I), in which inoculation was followed by treatment after six hours. However, the endophthalmitis was less violent than expected, a factor which may be explained by the spontaneous healing of wounds of the capsule in rabbits with the formation of only a partial cataract.

In the first experiments infection of the "needle track" in the cornea frequently occurred, followed by an infiltration of various extensions twenty-four hours later in control eyes and in eyes treated with sulfadiazine. When penicillin treatment was used infiltration was not seen. These observations suggest the effectiveness of penicillin therapy in pneumococcic corneal infections.

Penicillin was introduced by corneal bath and by iontophoresis. Despite the much higher concentration found in the aqueous after iontophoresis, no essential difference was noticed in the effect of the two forms of therapy except when the inflammation was very severe. The number of failures was about the same with the two types of treatment, but generally the eyes with the more advanced infection were treated iontophoretically with a 0.25 per cent solution and the eyes with the mildest infection with corneal baths.

Both corneal bath and iontophoresis have several disadvantages in clinical application. For iontophoresis some equipment is necessary. This treatment with the Birkhäuser or van Heuven electrode cannot be applied without some cooperation from the patient. Children, therefore, cannot be treated repeatedly. In very painful eyes the pressure exerted by the tubes is not easily tolerated. It is probable that other forms of application, especially the use of wetting agents combined with penicillin, will be more satisfactory in these respects than the ionization method and corneal bath. However, preliminary experiments with three wetting agents—zephiran (a mixture of high molecular alkyl, dimethyl and benzyl ammonium chlorides), aerosol IB (dibutyl sodium sulfosuccinate) and penetrasol B (a mixture of aerosol 1B, xylene, antipyrine and propylene glycol [38])—did not fulfil these expectations.

Further studies will show what concentrations of penicillin can be used in human eyes. In experiments on animals with severe infections the treatment

38. Herrmann, F.; Sulzberger, M. B., and Baer, R. L.: New Penetrating Vehicles and Their Solvents, Science **96**:451, 1942.

was continued twice daily even after an abrasion caused by previous iontophoretic treatments with 0.25 per cent solution had occurred. This epithelial damage healed in a few days without any residual opacity except in an eye in which a cloudy area remained in the center of the cornea for several weeks. The abrasions which occasionally were seen after repeated iontophoretic treatments with 0.1 per cent solution were small and healed in one day.

SUMMARY

1. Experimental intraocular infection caused by D. pneumoniae, type III and type X, was not stopped or was only temporarily improved by intensive local treatment with sodium sulfadiazine combined with sulfadiazine feeding when the treatment was initiated six hours after inoculation.

2. Severe intraocular infection caused by types III, X and VII was usually checked by local treatment with the sodium or the ammonium salt of penicillin in solutions of 0.25 per cent and 0.1 per cent even when this treatment did not start until twelve to thirteen hours after inoculation. The local applications were generally continued from two to four days.

3. Intraocular infection caused by the injection of types III and X with simultaneous injury of the lens capsule was treated successfully with penicillin in most instances in which the treatment was started six hours after injection and continued five to six days.

4. The iontophoretic introduction of the penicillin salt in 0.1 and 0.25 per cent solutions was occasionally more effective than the corneal bath (0.25 per cent solution) when the infection was very severe.

5. If repeated iontophoretic applications of 0.25 per cent solution were used because of the severity of the infection, corneal damage was noticed in the form of a large abrasion which usually healed without residual opacity after a few days.

6. Strains of types VI, XIV, XIX and XXIII showed in vitro the same sensitivity to penicillin as the strains of types III and VII used in the infection of eyes of rabbits.

Miss J. Di Grandi participated in all experiments and carried out the bacteriologic part of the study.

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COMBINED INTRACRANIAL AND ORBITAL OPERATION FOR RETINOBLASTOMA

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The purpose of this paper is to describe a new surgical approach for the treatment of retinoblastoma (glioma of the retina).

The results of enucleation of the eye in treatment of this tumor have been discouraging. An analysis of various series reported in the literature is shown in the accompanying rate table. The average survival rate even for short follow-up periods is only 18 per cent. If the reports of Hirschberg and Wintersteiner [1] are discounted as perhaps not in accord with the results of modern surgical technic, the statistics are somewhat less discouraging (a cure in 50 per cent of cases), although the most recent report of all is the least favorable (35 per cent of cures).

Results of Enucleation of the Eye for Retinoblastoma as Reported in the Literature

Date	Author	Follow-Up, Years	Total Cases	Cures	
				Number	Percentage
1868	Hirschberg [1a]	..	75	4	5
1897	Wintersteiner [1b]	2	497	65	13
1911	Adam [1c]	..	46	26	57
1916	Leber [1d]	..	28	16	57
1941	Parkhill and Benedict [1e]	1	31	11	35
	Grand total		677	122	18
	Total for last three		105	53	50

What is widely recognized as true but often minimized in the discussion of retinoblastoma is the tendency of the tumor to invade the optic nerve early and to extend backward within the confines of the nerve sheath to the intracranial cavity. Here it may spread into the chiasm and opposite optic nerve. It invades contiguous brain and meninges and spreads widely via the subarachnoid space.

It must be stressed that the extension of the tumor into the optic nerve is the really important consideration. For it is this process that goes on unseen, defying recognition by the routine methods of examination and preceding, as a rule, any extension through the sclera into orbital tissues and blood-borne metastasis to distant organs.

Read before the Section of Ophthalmology, New York Academy of Medicine, April 19, 1943.

From the Department of Surgery of the New York Hospital and Cornell University Medical College.

1. (*a*) Hirschberg, J.: Anatomisch Untersuchungen ueber Glioma Retinae, Arch. f. Ophth. **14**:30, 1868. (*b*) Wintersteiner, H.: Das Neuroepithelioma Retinae, Vienna, Franz Deuticke, 1897. (*c*) Adam, C.: Statistisches, Klinisches und Anatomisches ueber das Glioma Retinae, Ztschr. f. Augenh. **25**:330, 1911. (*d*) Leber, T.: Die Geschwulstbildungen der Netzhaut, in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, chap. 5. (*e*) Parkhill, E. M., and Benedict, W. L.: Gliomas of Retina: Histopathologic Study, Am. J. Ophth. **24**:1354, 1941.

In an admirable study of 119 cases in which the eye had been enucleated for glioma (retinoblastoma) from which there had been "no extra ocular extension," Reese [2] showed that in 63 (or 52 per cent) there had been invasion of the optic nerve posterior to the lamina cribrosa and in 51 the operative section was distal to the extension of the tumor. In other words, the operation failed to remove the tumor in 43 per cent of the 119 cases. The study showed further that the size of the growth in the globe had no bearing on the presence or degree of extension in the nerve, and sometimes a relatively small retinal tumor was accompanied by neural extension.

It has been twenty years since Jean,[3] writing about glioma of the retina (retinoblastoma), said, "It seems advisable that brain surgeons should attack these cases from within the brain [? skull] which they can do with reasonable safety." Yet since that time there have been reports of but two attempts at this approach to the treatment of retinoblastoma. It is the purpose of this presentation to reopen the discussion of the intracranial operation as part of a combined procedure for the cure of patients with retinoblastoma and to report 2 cases illustrating the advantages of the procedure.

Doubtless many patients having extension of the tumor into the optic nerve could be cured if enough of the nerve was resected in addition to enucleation of the eye. While it might be reasonably expected that sometimes nearly a centimeter of the nerve could be removed at the time of enucleation of the eye, actually the average length of the nerve found in Reese's study [2] was 3 mm. Some surgeons [4] have advised exenteration of the orbit so that more of the nerve may be resected when its section at the time of enucleation has passed through tumor. This more radical operation, besides being grossly disfiguring, would often fall short of giving the desired result when the nerve was invaded farther proximally.

Irradiation has been employed for some time in the treatment of the initial tumor and the residual tumor in the optic nerve following enucleation. The recent report by Martin and Reese [5] on the use of irradiation in the treatment of retinoblastoma is the most comprehensive statement to date on what can be expected from this form of treatment. These authors concluded that immediate enucleation of the eye is the safest plan if the tumor is unilateral. If both eyes are involved, with loss of vision, bilateral enucleation is the procedure of choice, although if any vision exists in one eye, they employ enucleation of the worse eye and irradiation of the other. The purposes for which irradiation is recommended are (1) treatment of early growths in an attempt to conserve vision in one eye and (2) treatment of postoperative residual or recurrent tumors.

Little can be expected from the irradiation of recurrent tumors that come to attention some time after enucleation of the eye. But the practice of employing irradiation promptly when microscopic examination of the enucleated eye reveals that tumor has invaded the optic nerve proximal to the point of operative section has much in its favor. Reese [2] and Martin and Reese [6] have stressed the value of this plan of treatment and established dosages for the maximum irradiation to

2. Reese, A. B.: Extension of Glioma (Retinoblastoma) into the Optic Nerve, Arch. Ophth. **5**:269 (Feb.) 1931.

3. Jean, G. W.: Discussion, Arch. Ophth. **51**:505, 1922.

4. de Schweinitz, G. E.: Diseases of the Eye, ed. 10, Philadelphia, W. B. Saunders Company, 1924, p. 536.

5. Martin, H. E., and Reese, A. B.: Treatment of Retinoblastoma (Retinal Glioma) Surgically and by Irradiation, Arch. Ophth. **27**:40 (Jan.) 1942.

6. Martin, H. E., and Reese, A. B.: Treatment of Retinal Gliomas by Fractionated or Divided Dose Principle of Roentgen Radiation: Preliminary Report, Arch. Ophth. **16**:733 (Nov.) 1936; footnote 5.

be employed. They recommended for both postoperative residual and recurrent growths the administration of 7,000 to 9,000 r over a three to four week period, followed by intraorbital implantation of radon seeds providing 5 to 10 millicuries. Their figures on a series of 6 cases show that if irradiation is instituted immediately after enucleation, when tumor is discovered in the remaining part of the resected optic nerve, the patient has a 66 per cent chance of cure. If growth of the tumor occurs after the initial course of therapy, no further irradiation can be given for fear of its causing necrosis; in fact, in 1 of their cases (case 20) fatal meningitis developed after exenteration of the orbit and implantation of radon seeds in the optic nerve.

While the results from enucleation of the eye combined with irradiation of the involved optic nerve stump are an improvement over those obtained from either enucleation or irradiation alone, there is justification for seeking methods that may provide still better results. It has not yet been made clear what the principal reasons are for failure in those patients who have had the combined treatment, but it may be surmised that many of the failures occur in those with neoplastic extension that has progressed to a point in the optic nerve where irradiation is not effective. From the beginning the obstacle in the path of cure of more patients with retinoblastoma has been the inability to remove surgically or affect adequately by irradiation that part of the invaded optic nerve that cannot be reached through the orbit.

The seemingly obvious adjunct to the routine procedures employed by the ophthalmologist is intracranial resection of the optic nerve. Jean [7] lays the blame on the ophthalmologist for not seeking the aid of the neurosurgeon, but perhaps the latter is as much to blame for not taking some initiative in the matter.

The first recorded use of intracranial resection of the optic nerve invaded by a retinoblastoma is in the case of Dott and Meighan.[8] The patient was a child of unspecified age in whom the tumor filled the enucleated eye though it did not traverse the sclera. But presumably tumor had been left in the optic nerve, since an "attempt" was made to remove more of the nerve through the orbit seven days after enucleation. Later radium was used, and still later the remainder of the optic nerve up to the chiasm was removed intracranially. "The region became infected but this later subsided," and the child's health was reported excellent one year later.

The other case of this kind is that reported by Rand [9] in 1934. In a girl of 4 years and 7 months enucleation of the eye was followed by local recurrence of the tumor in the orbit, but an intact conjunctiva covered the tumor anteriorly. Through an intracranial approach the orbit was unroofed, the nerve resected back to the chiasm and the intraorbital mass removed. Accidental disruption of the conjunctiva anteriorly provided a portal for infection and a cerebrospinal fluid fistula through the orbit was followed by meningitis and death.

Bennett [10] attempted avulsion of the optic nerve through the orbit, causing such trauma to the chiasm that postoperative hemianopsia developed in the remaining eye.

Perhaps these accounts afforded little incentive for other surgeons to attempt removal of the intracranial portion of the optic nerve, but it can hardly be said

7. Jean, G. W.: Glioma of the Retina, Correspondence, J. A. M. A. **100**:1793 (June 3) 1933.

8. Dott, N. M., and Meighan, S.: Intracranial Resection of the Optic Nerve in Glioma Retinae, Am. J. Ophth. **16**:59, 1933.

9. Rand, C. W.: Glioma of the Retina: Report of a Case with Intracranial Extension, Arch. Ophth. **11**:982 (June) 1934.

10. Bennett, H. P.: Case of Primary Intradural Tumor of the Optic Nerve, Brit. M. J. **1**:1041, 1905.

that the circumstances in any of the cases were ideal for a good result. The use of the intracranial operation in conjunction with enucleation of the eye should be carefully planned in such a way as to avoid the two chief threats to a successful outcome, namely, incomplete removal of the tumor and serious infection.

It is believed that the following 2 cases demonstrate a relatively safe and promising combined procedure for the total removal of the eye and its optic nerve.

CASE 1.—S. W., a 6 month old girl, on the recommendation of Dr. Irving J. Hausemann, was admitted to the New York Hospital in June 1942 because of recent convulsions. Her birth had been unremarkable and her development normal. The family history was not important. Some of the attacks were characterized by sudden turning of the head to the right, stiffening of all extremities and failure to respond. In other attacks the child would raise the arms over the head and become limp, so that the head would fall forward if she was held upright. At the same time she would turn the head aimlessly from side to side, stare vacantly and drool from the mouth. During these attacks, which lasted one to two minutes, there were no convulsive movements or cyanosis, and recovery was complete and prompt at the end of each attack. The attacks occurred from two to five times daily.

Examination revealed the baby to be healthy, with no abnormalities except a tumor of the right retina. Blood counts and chemical examinations of the blood gave normal results. The spinal fluid and roentgenograms of the skull were normal.

The right eye appeared to be normal in every way but for a rounded, elevated, grayish white mass in the retina to the temporal side of the optic disk. The mass was oval, apparently solid, well demarcated and slightly larger than the disk and had a fine capillary blood supply over its surface. The consensus of all the examiners, including several ophthalmologists and neurologists, was that the tumor was in all probability a retinoblastoma. Intracranial extension was considered a possible cause for the convulsions.

On July 10, with open mask ether anesthesia, a craniotomy was performed through a small, frontotemporal incision on the right. The brain and meninges exposed were normal in appearance. The right frontal lobe was elevated to expose the optic nerves and chiasm. These were normal in appearance, and that portion of the right optic nerve between the chiasm and the optic foramen was resected. The right optic foramen was plugged with a bit of muscle, and the wound was closed. But for moderate fever on the first two days the child's condition was excellent, and the postoperative course was uneventful.

The resected fragment of optic nerve measured 8 mm. and failed to show any abnormality on microscopic examination.

On July 23, with open mask ether anesthesia, the right eye was enucleated. The conjunctiva was incised, and the extraocular muscles were divided at their insertions into the globe. After being freed from Tenon's capsule, the eyeball, together with the remaining portion of the optic nerve (about 1.5 cm.), was lifted away with the enucleation spoon. The fascia was sutured posteriorly and Tenon's capsule sutured over a gold ball implant. The muscles were brought together with sutures in front of the implant, and the conjunctiva was closed.

The postoperative course was uneventful, and the child left the hospital in good condition ten days later.

Pathologic examination of the enucleated right eye showed an unusual type of glioma, which will be the subject of a separate report.

There is perhaps a point of debate about the exact nature of the retinal tumor in this case. In the six months since operation the child has had some convulsions, even with anticonvulsant medication, and epilepsy can definitely be diagnosed. The underlying cause may be tuberous sclerosis (epiloia), and the retinal tumor may be considered to have been related to this disease. However, the indications for enucleation of the eye had been definite enough in the beginning, and the two stage operation employed for total resection of the optic nerve, together with the removal of the eye, demonstrated the comparative ease and safety with which such operations can be performed.

CASE 2.—S. R., an 11 week old girl, referred by Dr. Bernard Samuels and the New York Eye and Ear Infirmary, was admitted to the New York Hospital Dec. 12, 1942. The child's birth and early development had been normal, but at the age of 6 weeks an unusual color of the right pupil was noted by the mother. A week later, when a physician was consulted, a diagnosis of retinal tumor of the right eye was made, and this was concurred in by all subsequent examiners.

Examination revealed the infant to be small, listless and somewhat undernourished looking. The head was moderately asymmetric and the scalp and face seborrheic. The abdomen was distended, and there was congenital deformity of some of the toes.

The right eye showed the characteristic changes due to a retinal tumor. The pupil gave a reddish yellow reflex, was dilated and did not react to light. The tumor filled the vitreous cavity, causing moderate secondary glaucoma and slight corneal edema. The left eye was normal.

After transfusion the child's condition seemed suitable for operation.

On December 15, with open mask ether anesthesia, a craniotomy was performed through a small, frontotemporal incision on the right side. The exposed meninges and brain were normal in appearance, as were the optic nerves and chiasm when seen after elevation of the frontal lobe. The intracranial portion of the optic nerve measured about 1 cm. from optic foramen to chiasm, and this portion was resected. The stump of the distal fragment of the optic nerve was coagulated with the electrosurgical unit and the foramen plugged with a piece of muscle. The wound was carefully closed. There was moderate fever for the first two days after operation, but thereafter the course was uneventful.

The resected fragment of nerve measured 1 cm. and failed to show any abnormality on microscopic examination.

On December 28, with open mask ether anesthesia, the right eye was enucleated. The conjunctiva was incised, and the extraocular muscles were divided at their insertions into the globe. After being freed from Tenon's capsule, the eyeball, together with the remaining optic nerve (about 1.5 cm.), was lifted away with the enucleation spoon. Posterior and anterior purse string sutures closed Tenon's capsule over a gold ball implant. The muscles were brought together with sutures in front of the implant, and the conjunctiva was closed.

Pathologic examination showed that the eye was filled with a typical retinoblastoma which reached, but did not extend along, the optic nerve.

While the microscopic examination of the optic nerve did not show that the tumor had invaded the optic nerve, there was no way of predicting this prior to operation. The child's general condition was none too good to begin with; nevertheless both operations were carried out with minimal reaction. There is little experience to go on in judging the ability of an infant without intracranial disease to withstand intracranial operation, but resection of the optic nerve, judging from this and the previous case, should be attended by little morbidity or risk to life.

THE OPERATION

The intracranial operation is performed through a comparatively small cutaneous incision, like that routinely employed by neurosurgeons in the "hypophysial approach." It begins just in front of the ear and curves upward and forward to end at or just in front of the hairline of the forehead (fig. 1, inset). The skin and galea are stripped from the temporal fascia and periosteum and retracted anteriorly. A small bone flap is made so that the anterior cut borders the supraorbital ridge as far laterally as possible, and the inferior cut is well down under the temporal muscle toward the zygoma. A little bone removed here with a rongeur after reflection of the flap facilitates the exposure. In infants the dura is adherent to the skull along the line of the coronal suture, which passes through the approximate center of the bone flap. Care must be taken, therefore, not to tear the dura or distort it unduly in elevating the bone.

The dura is opened by curving the incision just inside the anterior and temporal margins of the bony aperture. The posterior extent of the incision in the temporal region should traverse the middle meningeal artery, which requires coagulation or clipping. The frontal lobe is gradually retracted away from the floor of the frontal fossa, and the cisterna chiasmatis is evacuated. A good view of the optic nerve, chiasm and even the opposite nerve can be obtained without fear of damaging the brain, though special care must be taken to avoid injuring the olfactory nerves (fig. 1, *a*).

The arachnoid is stripped away from the optic nerve and a small nerve hook passed under to free it all around and isolate it from the adjacent carotid artery, which lies partly beneath and lateral to the nerve (fig. 1, *b*). The nerve is first transected at its junction with the chiasm. Then, while it is held on slight traction with a forceps, it is transected at the optic foramen. Neither of these sections is attended by more than slight vascular oozing, which can be controlled in the case of the chiasmal stump by light tamponade with a cotton pledget for a few moments. The ocular end of the nerve tends to retract into the foramen. Both the dural lining of the foramen and the nerve end are now coagulated by applying the electro-

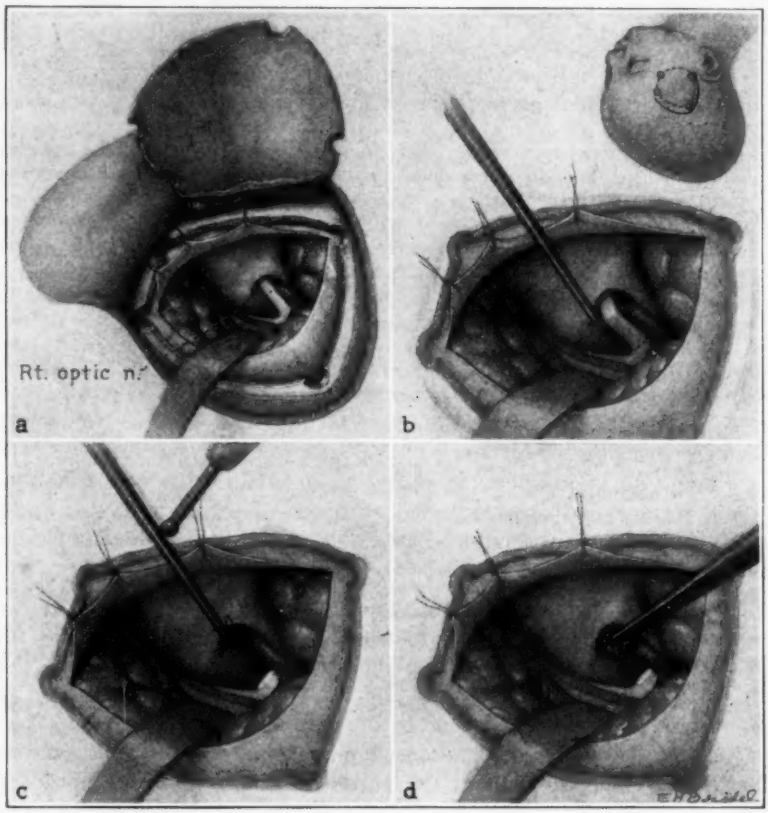

Fig. 1 (case 2).—Operative sketches showing the intracranial resection of the optic nerve: The optic nerves, chiasm and adjacent structures are exposed by elevation of the frontal lobe of the brain (*a*). The optic nerve is freed from the optic foramen to the chiasm (*b*). The inset shows the incision on the scalp and outline of the bone flap. After resection of the optic nerve, the stump of the remaining nerve is coagulated in the foramen (*c*). The optic foramen is plugged with a piece of muscle to prevent leakage of cerebrospinal fluid after enucleation of the eye with the remaining portion of the nerve (*d*).

surgical unit to a metal nerve hook passed into the foramen (fig. 1, *c*). A small piece of temporal muscle is firmly packed into the optic foramen to seal it off from the subarachnoid space (fig. 1, *d*). An alternative method is to turn down a lappet of dura to cover the foraminal opening, but this may be attended by some bleeding, which is to be avoided if possible.

Fig. 2 (case 2).—Eye and entire orbital portion of the optic nerve and nerve sheath removed in one piece.

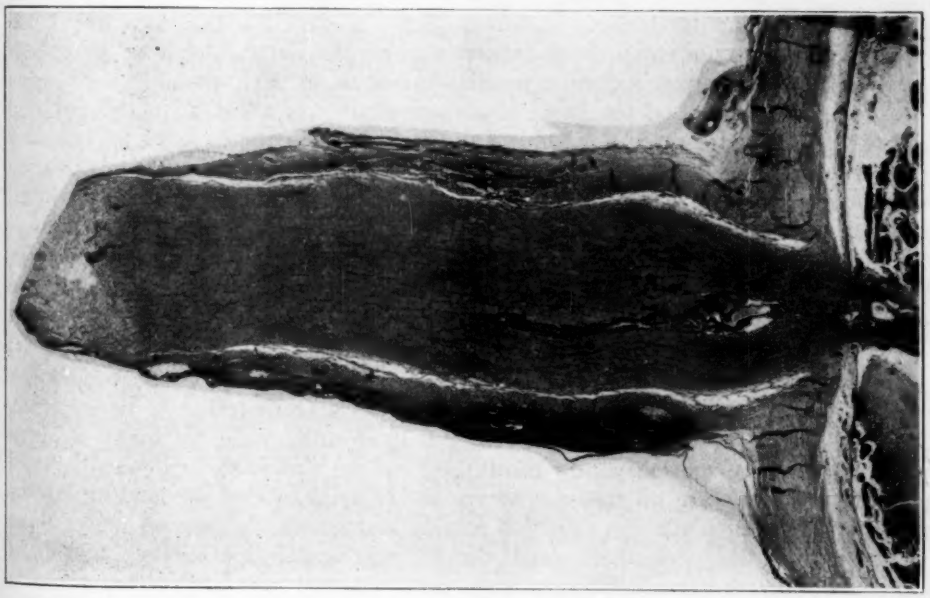

Fig. 3 (case 2).—Cross section of the optic nerve showing how completely the nerve and sheath are removed together.

In the closure, the dural margins are approximated and the replaced bone is held in position by one or two silk sutures passed through small drill holes. The temporal fascia, galea and skin are separately sutured with interrupted stitches of silk.

With infants and young children open mask ether anesthesia is preferable and an intravenous infusion of suitable fluids should be employed throughout and after the operation.

The secondary operation, enucleation of the eye with simultaneous removal of the remaining part of the nerve, has been carried out twelve and thirteen days later. It varies from the ordinary enucleation in a few particulars. All six extraocular muscles are tenotomized at their scleral insertions, but the optic nerve is not cut. Instead, an enucleation spoon is slipped behind the globe around the nerve, and the entire eye, optic nerve and nerve sheath are delivered intact by traction (fig. 2). Figure 3 shows how completely the optic nerve sheaths are removed with the nerve. Closure and prosthetic implantation are done in the usual manner. If too large a hole has been torn in Tenon's capsule posteriorly, a small purse string suture suffices to close it so that the implant will not slip out of position posteriorly.

COMMENT

The surgical procedure described is advocated in an effort to save that group of children who die from intracranial extension of retinoblastoma after inadequate treatment by enucleation of the eye. In spite of efforts to remove longer portions of the optic nerve by the orbital route, experience has shown that the operation of enucleation imposes limitations on the amount of nerve that can be resected. Reasons for avoiding exenteration of the orbit and for avoiding secondary intracranial operation have been given. The two choices remaining seem to be the procedure described here and that advocated by Martin and Reese. Experience with their method is still too limited for the drawing of sound, statistical conclusions. Since about half the eyes that are enucleated show extension down the nerve, and since immediate institution of heavy postoperative irradiation seems to save about two thirds of these, the expectation on the basis of present figures is about 80 to 85 per cent of cures. We cannot draw any statistical conclusions on the basis of our 2 cases. The relative merits of the two technics can be judged only when enough patients have been treated by each method and followed for a sufficient time thereafter. Some surgeons always prefer a radical surgical attack on malignant processes; others tend toward less radical surgical intervention plus irradiation. It might be added that in the treatment of retinoblastoma irradiation could still be employed after a radical surgical procedure, but the reverse would not be so safe or so satisfactory.

Perhaps the most important consideration in the proposed two stage operative removal of the eye and its entire nerve is the operative mortality to be expected. Doubtless until the contrary is proved it will be anticipated by many ophthalmologists that the mortality rate resulting from the procedure will outweigh the advantages. There are no statistics which are of assistance in predetermining the mortality rate, but it may be said that infants and young children on whom these operations will be performed stand simple intracranial operations, as well as successive operations, surprisingly well provided the necessary precautions in preoperative and postoperative care are employed. Two unnamed neurosurgeons cited by Jean [7] have stated that the risk of the intracranial resection of the optic nerve "should be very small," "2 per cent or thereabouts." We feel that the intra-

cranial operation should be attended by an operative mortality no greater than 5 per cent and probably considerably less than that.

It might be possible on some of the older children for one to perform both the intracranial and the orbital operation at one time, thus avoiding the longer hospital stay and the slightly greater danger of two anesthetics. This method was recommended by Rand. However, two short operations should be better withstood by an infant than one longer and more extensive. Also, the sealing off of the optic foramen would be more nearly assured if a little time was allowed to elapse before the orbital part of the nerve was removed.

The practice of performing the intracranial resection of the nerve first has certain advantages and disadvantages. The chief disadvantage is that in about 50 per cent of the cases the operation will have been performed needlessly, that is, in those in which it is eventually found that any extension of the tumor into the nerve could have been readily removed at the time of enucleation. But it is certain that the difficulties of the operation and the dangers to life are considerably increased if the intracranial operation is resorted to after it has been discovered that tumor remains in the optic nerve after enucleation of the eye. There is the distinct danger of causing meningitis if the orbital portion of the nerve is pulled back into the cranial cavity after a recent enucleation in which there has been potential if not frank contamination of orbital tissues. There is the possibility that the nerve, considerably thickened by tumor, cannot be pulled back through its canal. In this case, unroofing of the canal and orbit would greatly add to the dangers of the operation. Also, any delay between the primary enucleation and secondary intracranial operation would make extension of any remaining tumor from the nerve into adjacent orbital tissues, or farther toward the brain, more of a possibility than it would be if the procedures were reversed. It is believed that coagulation of the stump of the optic nerve in its canal and plugging of the optic foramen as carried out in the initial intracranial operation will prevent intracranial extension of the tumor. In addition, coagulation of the nerve end should for a time prevent local extension of the tumor within the orbit. In short, the intracranial resection of the optic nerve as the initial operation has everything to recommend it except that in about half the cases it will have been performed needlessly.

The advisability of enucleation of both eyes in the presence of bilateral retinal tumor is perhaps debatable, but if it should be resorted to, both nerves could be resected intracranially at one preliminary operation.

SUMMARY AND CONCLUSION

In retinoblastoma the most important consideration is the extension of the tumor into the optic nerve. It is believed that in nearly 50 per cent of the cases the standard operation of enucleation of the eye is inadequate. Exenteration of the orbit, or secondary intracranial resection of the optic nerve, is unsatisfactory. The prompt irradiation of residual tumor in the optic nerve after enucleation has improved the results; however, the best results of this treatment fall short of cure in at least 15 to 20 per cent of cases.

It is proposed that a combined, or two stage, intracranial and orbital removal of the eye and the entire optic nerve offers the greatest possibility of cure in cases of retinoblastoma. Two cases of retinal tumor are reported in which this method of treatment was employed.

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EXOPHTHALMOS DUE TO CHRONIC ORBITAL MYOSITIS

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The purpose of this paper is threefold: first, to call attention to a comparatively rare type of chronic inflammation of the extraocular muscles known as idiopathic or primary chronic orbital myositis; second, to suggest a simple test for its detection, and, third, to report 4 cases studied at the Institute of Ophthalmology, Presbyterian Hospital, New York.

By way of introduction it may be stated that chronic inflammatory or degenerative changes in the extraocular muscles have been reported in the following conditions: (1) syphilis of the orbit; (2) tuberculosis of the orbit; (3) myasthenia gravis, muscular dystrophies and degenerative myositis of fatty, amyloid, vascular or traumatic origin; (4) exophthalmic goiter, before and after thyroidectomy, and (5) apparent good health (idiopathic form).

Because of the rarity of syphilitic and of tuberculous orbital myositis and the ease with which the correct diagnosis can usually be made, no attempt will be made to discuss these forms here. The reader is referred to Offret,[1] who has reported in detail their clinical and pathologic characteristics. Orbital myositis associated with myasthenia gravis and the muscular dystrophies, together with the rare degenerative types (fatty, amyloid, vascular or traumatic), will also be excluded from consideration.

Reports of the histologic changes occurring in the extraocular muscles in exophthalmic goiter before thyroidectomy are rather few, but have been contributed by Moore,[2] Smelzer,[3] Benedict,[4] Wilson,[5] Dudgeon and Urquhart,[6] von Zalka,[7] Friedenwald[8] and others.[9] Most recent knowledge of such changes, however, has

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Read before the New York Academy of Medicine, Section of Ophthalmology, March 13, 1943.

1. Offret, G.: Les myositis orbitaires, Paris, Gaston Doin & Cie, 1939.

2. Moore, R. F.: Exophthalmos and Limitation of Eye Movements in Graves' Disease, Lancet **2**:701, 1920.

3. Smelzer, G. K.: A Comparative Study of Experimental and Clinical Exophthalmos, Am. J. Ophth. **20**:1189, 1937.

4. Benedict, W. L., and Knight, M. S.: Inflammatory Pseudotumor of the Orbit, Arch. Ophth. **52**:582 (Nov.) 1923.

5. Wilson, L. B., cited by Plummer, W. A., and Wilder, R. M.: Etiology of Exophthalmos, Arch. Ophth. **13**:833 (May) 1935.

6. Dudgeon, L. S., and Urquhart, A. L.: Lymphorrhages in the Muscles in Exophthalmic Goiter, Brain **49**:192, 1926.

7. von Zalka, E., cited by Thomas and Woods.[11]

8. Friedenwald, J. S.: Orbital Myositis and Choked Disc in Exophthalmic Goiter, Ann. Surg. **96**:995, 1932.

9. (*a*) Fisher, C., in discussion on Parker, W. R.: Double Luxation of Eyeballs in a Case of Exophthalmic Goiter, Tr. Am. Ophth. Soc. **19**:129, 1921. (*b*) Ellett, E. C.: Unilateral Exophthalmos, J. A. M. A. **116**:1 (Jan. 4) 1941. (*c*) Stallard, H. B.: Exophthalmic Ophthalmoplegia with Thyrotoxicosis, Brit. J. Ophth. **20**:612, 1936. (*d*) Kravitz, D., and Moehle, W. V.: Exophthalmos in Hyperthyroidism, Am. J. Ophth. **12**:527, 1941.

come from cases of so-called malignant exophthalmos, which has been well described by Burch,[10] Thomas and Woods,[11] Naffziger,[12] Ellett,[9b] Smelzer [3] and others.[13]

When one eliminates from discussion all cases of chronic orbital myositis of known cause there remains a small group of cases in which the cause is not known and the condition therefore merits the name of idiopathic, or primary, chronic orbital myositis.

IDIOPATHIC CHRONIC ORBITAL MYOSITIS

Chronic orbital myositis of the primary type is a comparatively rare condition of the extraocular muscles of unknown cause. It is characterized histologically by lymphocytic infiltration, fibrosis and degeneration of muscle fibers. Grossly, the muscles are pale, enlarged and cartilaginous, and they cut with a gritty feeling. Clinically, it produces edema of the lids, exophthalmos and limitation of ocular motility.

This clinical and pathologic picture has been reported a number of times in the literature under a variety of names, for example, idiopathic myositis,[14] orbital myositis,[1] Zenker's waxy degeneration,[1] pseudotumor [4] and exophthalmic ophthalmoplegia.[15] Most case reports, however, have been published under the title of chronic orbital myositis (French), pseudotumor (German, English and American) or exophthalmic ophthalmoplegia (English).

According to Michail and Rusu,[16] the first case report in the literature was made by Busse and Hochheim in 1903. These observers failed to note that Gleason,[14] of this country, reported a case in the same year, fully describing the clinical and histologic observations. Since then a number of case reports, mostly in the French and German literature, have been added, generally under the title of pseudotumor. In 1909 Birch-Hirschfeld [17] reviewed the literature on pseudotumor and added some case reports of his own. In 1923 Benedict and Knight [4] were able to find only 5 additional case reports of pseudotumor, all of which were in the foreign literature.

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10. Burch, F. E.: Exophthalmos of Graves' Disease, Minnesota Med. **12**:668, 1929.

11. Thomas, H. M., and Woods, A. C.: Progressive Exophthalmos Following Thyroidectomy, Bull. Johns Hopkins Hosp. **59**:99, 1936.

12. Naffziger, H. C.: Progressive Exophthalmos Associated with Disorders of the Thyroid Gland, Ann. Surg. **108**:529, 1938.

13. (*a*) Merrill, H. G., and Oaks, L. W.: Extreme Bilateral Exophthalmos, Am. J. Ophth. **16**:231, 1933. (*b*) Levy, A. H.: Bilateral Exophthalmos, Proc. Roy. Soc. Med. **24**:454, 1931. (*c*) Frazier, C. H.: Unilateral Exophthalmos: A Clinical Report of Five Cases, S. Clin. North America **3**:281, 1923. (*d*) Niall, F.: Persistent and Progressive Exophthalmos: Relationship to Graves' Disease, Tr. Ophth. Soc. Australia **1**:56, 1940. (*e*) Kistner, F. B.: Decompression for Exophthalmos (Two Cases of Naffziger), J. A. M. A. **112**:37 (Jan. 7) 1939. (*f*) Zimmerman, L. M.: Exophthalmos Following Operation for Relief of Hyperthyroidism, Am. J. M. Sc. **178**:92, 1929. (*g*) Rosenbaum, J.: Malignant Exophthalmos, Canad. M. A. J. **36**:12, 1937. (*h*) Stewens, H.: Progredienter Exophthalmus nach Basedow-operation: Ueber Beziehungen zwischen Thyreoidea und Hypophyse, Ztschr. f. Augenh. **75**:137, 1931. (*i*) Ginsberg, S.: Postoperative Progressive Exophthalmos with Low Metabolic Rate, Ann. Int. Med. **13**:424, 1939.

14. Gleason, J. E.: Idiopathic Myositis Involving the Extraocular Muscles, Ophth. Rec. **12**:471, 1903.

15. Brain, W. R., and Turnbull, H. M.: Exophthalmic Ophthalmoplegia, Quart. J. Med. **7**:293, 1938.

16. Michail, D., and Rusu, L.: Les myosites des muscles périoculaires, Ann. d'ocul. **177**:97, 1940.

17. Birch-Hirschfeld, A.: Die Erkrankungen der Orbita, entzündliche Pseudotumoren, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 3, p. 51; Diseases of the Orbit, in Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1930, vol. 9, pt. 1, chap. 13.

At that time they reported 6 cases, in 2 of which there was chronic nonspecific myositis. In 1925 Orlow [18] collected 4 more case reports and added 1 of his own. He insisted that this type of myositis was distinct from syphilitic or tuberculous myositis and that it should be placed in a class by itself. In 1938, under the title of exophthalmic ophthalmoplegia, Brain and Turnbull [15] reported their findings in a series of 31 cases, in some of which there were signs of hyperthyroidism. In 1939 Offret,[1] who had had some unhappy experiences with this condition (1 of his patients was a physician), published an excellent monograph on the subject. He had been able to collect and report on 34 cases, of which the myositis was of proved tuberculous origin in 6, of syphilitic origin in 5 and of unknown cause in 23.[19] In 1940 Michail and Rusu [16] reviewed 11 reported cases and added 2 of their own. In the same year Niall [13d] presented an excellent paper on this subject and reported 11 cases (5 in detail), in some of which there was evidence of hyperthyroidism. Others who have contributed to this subject are Reese,[20] Samuels,[21] O'Brien and Leinfelder,[22] Williamson-Noble,[23] Colley,[24] Ellett,[9b] Dalsgaard-Nielsen,[25] Schmencke,[26] Hine,[27] Franceschetti and Rutishauser,[28] Sautter [29] and others.[30]

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18. Orlow, K.: Zur Pathogenese der Pseudotumoren der Orbitos: Myositis fibrosa der auseren Muskeln der beiden Augen, Monatschr. f. Augenh. **39**:460, 1925.

19. Of these 23 cases, there had been microscopic studies in only 14 to prove the presence of myositis. In the remaining 9 cases the clinical picture of a tumor of the orbit was presented but at operation no tumor but only chronic inflammation was found. Had a biopsy specimen of the extraocular muscles been taken at the time of operation, the presence of orbital myositis might have been proved in many of these cases also.

20. Reese, A. B.: Etiology of Exophthalmos, Tr. Am. Acad. Ophth. **39**:65, 1934; Unilateral Exophthalmos and Its Surgical Treatment, Pennsylvania M. J. **43**:605, 1940; Exophthalmos: Its Pathology and Ocular Manifestations, New York State J. Med. **33**:73, 1933.

21. Samuels, B.: Some Notes on Orbital Tumors, Arch. Ophth. **7**:868 (June) 1932.

22. O'Brien, C. S., and Leinfelder, P. J.: Unilateral Exophthalmos, Am. J. Ophth. **18**:125, 1925.

23. Williamson-Noble, F. A.: Inflammatory Pseudotumor of the Orbit, Brit. J. Ophth. **10**:65, 1926.

24. Colley, T.: Inflammatory Pseudotumor of the Orbit: Case Report, Brit. J. Ophth. **19**:93, 1935.

25. Dalsgaard-Nielsen, E.: Pseudotumor orbitae, Acta ophth. **17**:418, 1939.

26. Schmencke, A.: Beitrag für Histologie de "entzündlichen Pseudotumor," Klin. Monatsbl. f. Augenh. **76**:207, 1926.

27. Hine, M. L.: An Extra-Dural Tumor of the Optic Nerve, Tr. Ophth. Soc. U. Kingdom **42**:181, 1922.

28. Franceschetti, A., and Rutishauser, E.: Die entzündlichen Pseudotumoren der Orbita, Arch. f. Ophth. **137**:93, 1937.

29. Sautter, H.: Pseudotumor der Orbita, Klin. Monatsbl. f. Augenh. **100**:29, 1938.

30. During a cursory review of the literature on exophthalmos, it soon became obvious that a number of writers may have reported this condition under other titles, thus making a complete review of the subject almost impossible. Then, too, a number of observers gave excellent clinical and gross pathologic reports but failed to describe the histologic character of the extraocular muscles, thus making it impossible for us to learn whether chronic myositis was present. Abstracted material was used when the original articles were not available. This fact may account for our failure to find pathologic reports in some cases. (*a*) Pollems, W.: Ueber tumorförmige Amyloidosis in der Orbita, Arch. f. Ophth. **101**:346, 1920. (*b*) Pines, B.: Ein Fall von beiderseitiger entzündlicher Geschwulst der Augenhohle (Pseudotumor), Klin. oczna **6**:116, 1928; abstracted, Zentralbl. f. d. ges. Ophth. **21**:117, 1929. (c) Marbaix, and Van Duyse, D.: Pseudoblastome de l'orbite, Arch. d'opht. **39**:466, 1921. (*d*) Imanishi, T.: Ueber die entzündliche lymphomatose der Orbita, abstracted, Klin. Monatsbl. f. Augenh. **70**: 281, 1922. (*e*) Huber, O.: Ein Fall von entzündlichen Pseudotumor der Orbita, ibid. **65**: 718, 1920. (*f*) Tauferová-Karásková, J.: Inflammatory Pseudotumors of Orbit, abstracted, Zentralbl. f. d. ges. Ophth. **26**:827, 1932. (*g*) Seka, W. A.: Pseudotumor der Orbita, abstracted,

Of the three names used to describe the disease (chronic orbital myositis, exophthalmic ophthalmoplegia and pseudotumor), chronic orbital myositis is undoubtedly the best, because it represents a definite pathologic and histologic entity. The clinical term exophthalmic ophthalmoplegia was coined by Brain because he believed that the extreme swelling of the tissues of the orbit produced both the exophthalmos and the ophthalmoplegia. Pseudotumor is perhaps the poorest term, because it denotes so many different pathologic conditions. It was first used by Birch-Hirschfeld [17] to describe exophthalmos in which no tumor was demonstrable. He classified cases of pseudotumor into three groups:

Group I consisted of cases in which the cardinal signs of orbital tumor (exophthalmos, displacement and disturbed motility of the globe) were present but the exophthalmos was cured by medical means, such as use of potassium iodide, mercury or quinine. The exophthalmos in these cases was supposed to be of syphilitic, tuberculous or hematogenous causation in spite of the fact that all known tests failed to substantiate the presence of any of the conditions suspected.

Group II included cases of exophthalmos in which the clinical diagnosis was orbital tumor but in which no tumor was found at operation. This group must have included some cases of exophthalmos due to thyroid disease, varicose veins of the orbit, diffuse lymphoblastoma, and so on.

Group III comprised cases of exophthalmos in which an operation revealed an abnormal orbital mass which on histologic study proved to be a chronic inflammatory process of a nonspecific nature containing diffuse follicular areas. A review of the case reports of this group indicates that it should be subdivided into (1) cases

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in which the extraocular muscles were chiefly or primarily involved and (2) those in which the muscles were spared and only the orbital fat was affected.[31]

From this classification it is obvious that the term pseudotumor represents a number of different pathologic conditions, and that this diagnosis only covers up ignorance of the exact pathologic nature of the disease process in the orbit. This term should be employed only as a clinical diagnosis, and then only when a histologic study of the orbital pathologic changes is impossible. When microscopic material is available for study, a more specific histologic diagnosis should be made. Such histologic studies must be forthcoming if the confusion surrounding this diagnosis is to be clarified.

REPORT OF CASES

CASE 1.—In March 1936 Mrs. R. D., a housewife aged 37, came to the Institute of Ophthalmology complaining of exophthalmos on the left side and diplopia of seven months' duration. She stated that the onset of her symptoms had been gradual and that for a time she had been able to overcome her diplopia by tilting her head backward. Later it had become necessary to keep the left eye covered to avoid diplopia and nausea. She had noticed no pain, visual impairment, edema or inflammation about her eyes except for swelling of the left eye following a bump over the left temple six months before she entered the hospital. Some time after the accident her left eye began to protrude.

Her past medical history was essentially irrelevant. There was no history of hyperthyroidism. After the birth of her daughter fifteen years before she had been told she had

Fig. 1 (case 1).—The eyes before operation. The patient was looking straight ahead with the right eye fixing.

nephritis and advised not to become pregnant again. Six years and again four years before she had been delivered of a stillborn infant and had subsequently suffered from nephritis and edema of the legs.

Examination showed her vision to be 20/20 in the right and 20/30 in the left eye. Her lids, conjunctivas, lacrimal sacs, intraocular tension and fundi were essentially normal. A slight ptosis was noted in the left eye. This eye was directed downward 15 degrees of arc and outward 5 degrees of arc. The patient was unable to elevate it above the horizontal plane. Readings on the exophthalmometer were 18 mm. for the right and 20 mm. for the left eye (fig. 1).

A report from her physician stated that the results of all her examinations and tests had been negative. In the hospital a routine urinalysis gave negative results, as did a Wassermann test of the blood. The spinal fluid chemistry and cell count were normal. The colloidal gold curve was normal. General physical and neurologic examination disclosed nothing significant. A roentgenogram of the left orbit showed increased soft tissue density and slight evidence of increased intraorbital pressure. The basal metabolic rate was + 4 per cent.

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31. There may be no justification for dividing Birch-Hirschfeld's third group into two subgroups, since the nature of the tissue change is histologically the same whether the orbital fat or the extraocular muscles are involved. A number of cases of pseudotumor have been reported, however, in which no mention was made of an abnormal state of the extraocular muscles or of their histologic character. Failure of the surgeon to remove a biopsy specimen of the orbital muscles along with the tumor mass found in the orbit may account for the apparent lack of muscle changes in such cases. It is therefore possible that in all cases of Birch-Hirschfeld's third group of pseudotumor, the orbital muscles were involved at least to some extent.

An intraorbital neoplasm was suspected and an exploratory operation of the left orbit through a lateral canthotomy done. The upper part of the orbit was normal, but below the globe a tumor was found running along the floor of the orbit from the globe to the apex. This growth seemed to infiltrate the inferior rectus muscle and was loosely attached to the periosteum of the orbital floor. It was about the size of a walnut, was fibrous and seemed to be encapsulated posteriorly. The surgeon noted that "undoubtedly by its check action on the inferior rectus muscle the tumor limited upward rotation of the globe." An attempt was made to remove the growth and at the same time save the inferior rectus muscle. The growth involved the muscle so intimately, however, that it was necessary to remove the muscle along with the tumor. Healing occurred without complications, and the patient left the hospital shortly. Follow-up examination four years later showed that the patient still had diplopia; the left pupil was dilated and reacted sluggishly to light. All movements of the extraocular muscles were limited, especially nasalward and downward.

Fig. 2 (case 1).—Lymphocytes, fibrosis and muscle fiber degeneration (× 200).

The pathologic report stated that the process consisted of chronic inflammation with degeneration of the muscle fibers. The histologic diagnosis was chronic inflammation and idiopathic myositis, or Zenker's waxy degeneration (fig. 2).

CASE 2.—In July 1939, I. M., aged 55, a merchant, presented himself for examination because of diplopia and inability to elevate his right eye (fig. 3*A*). His difficulty had begun seven months before with diplopia and turning downward of the right eye. He complained of no pain or inflammation in his eyes. His vision was good in each eye, but in order to see straight ahead with the right eye he had to tilt his head backward. His general health was good.

The patient's history disclosed chronic sinus disease, for which he had had the middle turbinate bone removed in 1916 and an operation on the frontal sinus in 1919. In 1933 he had an attack of benzene poisoning and a spontaneous pneumothorax of the right lung which was considered to be of unknown causation. This cleared up spontaneously in a few months.

A number of roentgenograms of the chest taken at that time showed an old bilateral healed pulmonary tuberculosis with thick fibrosis and small calcifications in the apex of the upper lobe of the right lung. Roentgenograms of the chest taken yearly to the time of writing have not changed this diagnosis and have shown no recent infiltration of either pulmonary field. There was no history of thyroid disease.

Two of the patient's brothers were said to have retinitis pigmentosa, and the son, aged 24, was said to have bilateral central scotomas and optic nerve atrophy due to macular changes, perhaps of a tuberculous inflammatory process.[32]

Examination showed the patient's vision to be 20/20 — 3 in the right eye and 20/20 — 2 in the left with a correction of + 1.00 D. sph. ⌒ 0.50 D. cyl., axis 100 before each eye. The lids, conjunctivas, lacrimal sacs and fundi were essentially normal. Examination of the extraocular muscles gave a first impression of a paralysis of elevation associated with exophthalmos on the right side. All movements of the left eye appeared normal. The right eye was turned directly downward about 25 degrees of arc, and the patient was unable to elevate the globe even so much as 5 degrees from this fixed position. Closer examination of the rotations of the right eye showed that all the extraocular muscles of this eye were active except the superior rectus muscle. The inferior oblique muscle was unable to elevate the fixed globe, but it was able to rotate the globe outward when the patient was asked to look up and to the left. The levator function was intact, and no ptosis was present; in fact, there

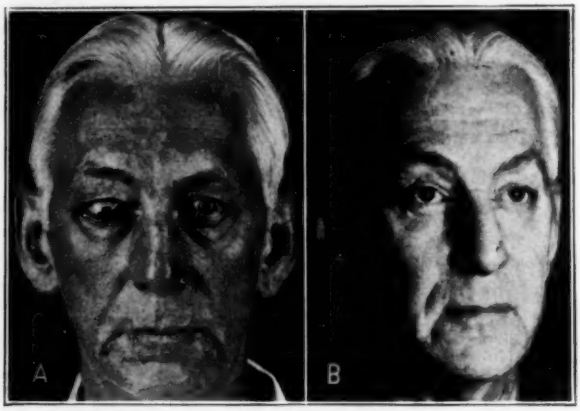

Fig. 3 (case 2).—*A*, the patient before operation, looking straight ahead with the left eye fixing. *B*, the patient after operation, looking straight ahead with both eyes fixing.

seemed to be an overaction of the right levator muscle, which accentuated the apparent exophthalmos and produced a frightened staring expression (fig. 3 *A*).

Because the observations on the muscles did not correspond with those in most cases of paralysis of the superior rectus muscle, some inflammatory process binding the globe down to the floor of the orbit was suspected. A wisp of cotton saturated with 10 per cent cocaine hydrochloride was inserted in the inferior cul-de-sac to effect deep local anesthesia. A fixation forceps was then applied to the tendon of the inferior rectus muscle and gentle traction exerted to rotate the globe upward. This met with firm resistance, and it was impossible to elevate the globe even so much as 10 degrees of arc. This condition was thought to be due to some inflammatory or neoplastic process binding the globe or the inferior rectus muscle down to the floor of the orbit.

Since the right eye was fixed in a downward position of gaze, it was necessary to have the patient tilt his chin upward until the two corneas were on the same plane in order to get the exophthalmometer readings. These were 15 mm. for each eye. Had it been possible to take these measurements in the usual manner, there is little doubt that an exophthalmos of 2 or 3 mm. at least would have been recorded.

A general physical examination revealed only vitiligo of the face and both arms. The Mantoux test gave a reaction of 2 plus, and a roentgenogram of the chest showed bilateral healed pulmonary tuberculosis with calcification and fibrosis in the apex of the right lung. The basal metabolic rate was + 16. The Wassermann reaction of the blood was negative;

32. Eagleton, W. P.: Personal communication to the authors.

a routine blood count and differential count were normal, and routine urinalysis gave negative results. A roentgenogram of the orbits and optic canals revealed only a slight increase in soft tissue density of the right orbit, with nothing to indicate its cause.

Because it was thought that some chronic inflammatory or neoplastic process was binding the globe down to the floor of the orbit, the orbit was explored through a lateral canthotomy in August 1939. A solid, fibrous, smooth, firm, dark bluish mass below the globe, which seemed to be attached to all sides of the inferior rectus muscle, was observed. The mass began 5 or 6 mm. from the insertion of the inferior rectus muscle to the globe and extended to the apex of the orbit. The growth was estimated to be 30 mm. long, 8 to 10 mm. thick and 10 to 12 mm. wide. It cut with difficulty with scissors and was removed in five pieces. We wished to save as much of the inferior rectus muscle as possible. But no normal tissue of this muscle was noted, and at the conclusion of the operation almost all of the tumor

Fig. 4 (case 2).—Lymphocytes, fibrosis and muscle fiber degeneration (× 200).

mass and the inferior rectus muscle had been removed. The optic nerve, floor of the orbit, globe and the other rectus muscles appeared to be normal to palpation. At the conclusion of the operation the globe was freely movable. The wound was closed without drainage. The patient made an uneventful recovery.

Since the operation the patient has been able to move his right eye moderately from side to side but not below the horizontal plane. With his head tilted forward about 10 degrees of arc he has single binocular vision, and when he looks downward, the right upper lid covers the pupil so that he does not see double. His vision is 20/20 in each eye, and his fundi and pupillary reactions are normal. The patient does not wear an occluder now and asserts that he is more comfortable than before operation (fig. 3*B*).

Dr. A. B. Reese's pathologic report stated: "The specimens contained considerable connective tissue and degenerated striated muscle fibers which were undergoing fibrosis. Scattered through the tissue were numerous irregular focal areas of inflammation containing large numbers of lymphocytes and a few plasma cells. Within and surrounding these inflammatory areas there were degeneration and destruction of muscle tissue; some of the muscle fibers

showed swelling and loss of striae. In some regions inflammatory cells were closely packed together in almost solid masses. In other areas there was a looser arrangement. The process in all of these sections was essentially one of muscle fiber degeneration and fibrosis accompanied by an inflammatory process" (fig. 4).

Dr. V. Kneeland Frantz studied the sections and reported as follows: "I could not see any evidence of tuberculosis in these sections. The lesion appeared to be a chronic inflammatory one, and I would suspect that the degeneration of muscle was a secondary inflammation. There was no evidence of tumor."

The microscopic diagnosis was chronic orbital myositis, or Zenker's waxy degeneration.

CASE 3.—B. G., aged 51, a machine operator, came to the Vanderbilt Clinic in October 1940, complaining of diplopia of six weeks' duration (fig. 5 *A*).

He stated that his present illness had begun six months previously with a swelling of the left lower lid. This was not associated with redness, pain or discharge. Three months later a similar swelling appeared in the left supraorbital region. This increased slowly until the time of his visit to the clinic. He had noticed no loss of vision in either eye, no pain and no inflammation and knew of no injury to either eye. His general health had always been good, and there was no history of thyroid disease.

Examination of the eyes showed the vision to be 20/30 in the right and 20/30—2 in the left. The lids, conjunctivas and lacrimal sacs were normal. The intraocular tension was 13 mm. (Schiøtz) in each eye. The exophthalmometer readings were 17 mm. for the right

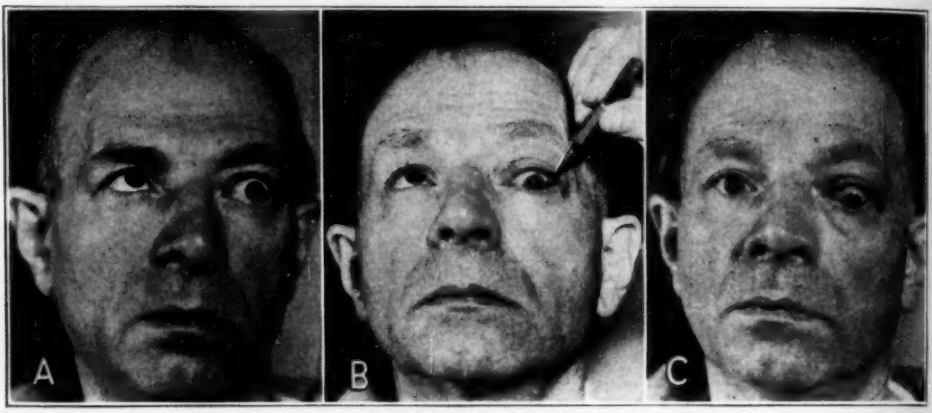

Fig. 5 (case 3).—*A*, the patient before operation, looking up with the right eye fixing. *B*, a traction test, which failed to elevate the left eye. *C*, the patient after operation, looking straight ahead with the right eye fixing.

and 21 mm. for the left eye. It was impossible to replace the left eye in the orbit by pressure backward. No lagophthalmos, no bruit and no pulsation were present. The pupillary reactions, fundi, perimetric fields and blindspots were normal. Clinical microscopy revealed nothing abnormal.

Examination of the extraocular muscles showed the movements of the right eye to be normal. The left eye was directed downward 15 to 20 prism diopters and slightly outward. The patient was unable to elevate the left eye above the horizontal plane (fig. 5 *A*).

A provisional diagnosis of intraorbital tumor associated with paralysis of the left superior rectus muscle was made and the patient sent to the hospital. While there he had a thorough clinical and laboratory study. A general physical, a urologic and a neurologic examination revealed nothing significant.

A roentgenogram of the skull, orbits and optic canals was essentially normal except that it showed some areas of porosity of the bones of the skull suggestive of parathyroid dysfunction and several areas of diminished density suggestive of metastasis. Roentgenograms of the long bones, chest, gastrointestinal tract, feet and hands did not reveal neoplasm, metastasis or sarcoid.

Because of the porosity of the bones suggestive of hyperparathyroidism, studies of the blood chemistry were made. These gave normal results except that on one occasion the protein level was 8.9 mg. per hundred cubic centimeters; on a second examination the value was found to be 7 mg. The normal blood chemistry ruled out hyperparathyroidism.

The reaction to a tuberculin test with 1:100,000 dilution was negative after forty-eight hours, but with a 1:10,000 dilution the reaction was graded 2 plus.

Since the clinical and laboratory tests did not reveal the nature of the disease, an orbital exploratory operation was recommended. Before operation two different attempts were made to elevate the globe mechanically by means of traction on a tissue forceps attached to the tendon of the inferior rectus muscle (fig. 5 *B*). These failed to elevate the globe on two different occasions and led to a preoperative diagnosis of chronic orbital myositis of the inferior rectus muscle.

At the time of operation a lateral canthotomy was done and the wound enlarged downward to expose the orbital contents below the globe. Palpation revealed a firm mass situated below the globe and attached to the inferior oblique muscle. This extended along the floor of the orbit toward the apex and seemed to be loosely adherent to the periosteum of the floor. The mass was removed and the globe freed of its extraneous attachments. At the conclusion of the operation the globe was more freely movable with traction in all directions. The wound was closed without drainage and the patient discharged from the hospital on the fifth postoperative day.

The wound healed promptly but the left eye returned to its position of downward rotation. Ten weeks after the operation the right eye was essentially normal, while the left presented exophthalmos of 2 to 3 mm., fixation in downward rotation of about 30 degrees of arc (fig. 5 *C*) and movement of the globe of about 10 degrees in abduction, 8 degrees in adduction and about 5 degrees in elevation and depression from its fixed position of downward gaze. The upper lid of the left eye moved freely up and down synchronously with the right when the right eye was fixing; with the left eye fixing there was a marked overaction of the levator muscle on the same side, with pronounced contralateral overaction of the right superior rectus and levator muscles. When both eyes were directed downward about 30 degrees of arc, the upper lid of each eye was at the upper part of the limbus, indicating no overaction or spasm of Müller's muscle or of the levator muscle in this position. A firm, immovable, painless, slightly nodular mass could be palpated through the lower fornix in the lower temporal quadrant in the orbit along the orbital margin between the floor and the globe. There were slight edema and fulness of the upper lid. Examinations of the exterior and interior of the eyes otherwise gave normal results.

A second exploratory operation through the external canthus and the inferior cul-de-sac exposed a firm, nodular mass of fibrous consistency wedged between the globe and the floor of the orbit in the lower temporal quadrant. This mass was dissected free from the floor and the globe. It extended backward along the inferior rectus muscle toward the apex of the orbit. An attempt was made to dissect it free from the inferior rectus muscle, but at the conclusion of the operation this muscle had been reduced to a narrow inelastic fibrous band about 6 mm. wide anteriorly. A soft mass, presumably the posterior end of the enlarged inferior rectus muscle, was palpated deep in the orbit but was not removed because of its inaccessibility and because of the danger to the deeper orbital vessels and nerves. The wound was closed without drainage. At the conclusion of the operation the eye was movable in all directions of gaze.

One month later the left eye had returned to its original position of downward gaze. The patient complained bitterly of diplopia and the appearance of his left eye. A series of deep roentgen ray treatments were given without any apparent effect. With the hope of improving the patient's appearance a third operation was performed five and one-half months after the first exploration. At this time the adhesions between the globe and the floor were freed and the superior rectus muscle resected to elevate the globe to the primary position.

One year after the first operation there was no exophthalmos present, but a slight edema of the upper lid was still noticeable. The globe was fixed in the primary position, from which the patient could rotate his eye about 10 degrees to the right and to the left. Neither elevation nor depression was present, and a firm, cartilaginous mass could be palpated through the skin of the lower lid between the globe and the inferior orbital margin. Six months later the left eye once more had rotated downward about 20 degrees of arc, where it was fixed and could not be elevated or depressed. Diplopia was present, though less annoying, and the patient was gradually adjusting himself to his disability and appearance.

The laboratory report by Dr. John S. McGavic was detailed and interesting. Microscopic examination showed that the specimen consisted of muscle, nerve, fat and fibrous connective tissue (fig. 6). About one half of the tissue was striated muscle; parts of this were essentially normal in appearance but a good many of the muscle fibers showed varying degrees of degenerative change. This change was characterized by pale staining, partial loss of cross striations and a granular appearance instead of the usual cleancut appearance of normal

striated muscle. There was an unusually large amount of fibrous tissue, which contained relatively few nuclei and which was somewhat more refractile than usual. In one place there was a localized collection of lymphocytes, but this was the only evidence of an inflammatory reaction. An unusually large amount of nerve tissue was present, but the arrangement and the absence of pathologic changes in this tissue seemed to nullify its importance. In summary, these sections showed only slight degenerative changes in striated muscle. The changes were similar to, but less marked, than those in cases 1 and 2, in which, as in this case, a diagnosis of Zenker's waxy degeneration was made.

Dr. A. P. Stout said: "This is a fragment of striated muscle which shows evidence of degenerative change, represented by fragmentation, vacuolation, loss of cross striation of the muscle fibers and some proliferation of fibrous tissue between and around the individual muscle fibers. . . . Adjacent to the striated muscle is a rather dense mass of fibrous tissue which is peculiar in that mixed up with it are what appear to be many elastic fibers and

Fig. 6 (case 3).—Lymphocytes, fibrosis and muscle fiber degeneration (× 200).

what appear in the trichrome stain to be stray bundles of smooth muscle. It is not a neoplasm and represents some sort of degenerative process affecting striated muscle with which I am not familiar."

Dr. Fred Stewart, of the Memorial Hospital for the Treatment of Cancer and Allied Diseases, felt that this was a case of Zenker's waxy degeneration.

Case 4.—J. K., aged 71, a farmer, came to the Institute of Ophthalmology in November 1940, because his left eye "felt stiff and sore in the morning." In spite of the fact that he had definite limitation of upward movement and exophthalmos of the left eye, he did not complain of diplopia. The present trouble had begun six months previously, after he had sprayed his trees. His general health had always been good, and he gave no history of thyroid disease.

Examination of the eyes showed that the vision was 20/25 in the right and 20/30 in the left without glasses. The lids, conjunctivas, lacrimal sacs, intraocular tension, peripheral fields, blindspots and fundi were normal. The right pupil measured 4 mm. and the left 2 mm.

There was slight limitation of upward movement in the left eye. The Hertel exophthalmometer gave readings of 15 and 19.

A general physical examination gave negative results. The Wassermann reaction of the blood and the sedimentation rate were normal. A routine urinalysis, red blood cell count and differential count gave normal results. An examination of the sinuses failed to reveal anything abnormal, and a roentgenogram of the head, showing the optic canals and orbits, disclosed only a slight increase in soft tissue density of the left orbit.

In five weeks the patient's exophthalmos progressed, disturbing diplopia developed and the upward excursion of the left eye became almost nil. The patient was sent to the hospital and the left orbit explored through a lateral canthotomy. The exploration revealed the presence of a large firm mass extending from the posterior surface of the globe backward along the floor to the apex of the orbit. A biopsy specimen was taken of the tumor mass and the wound closed without drainage. At the time of discharge from the hospital, five days later, the globe was once more in the primary position. One year later the patient was able to carry on his work as a farmer without complaint.

The histologic report of Dr. John S. McGavic was as follows: "The specimen consisted of muscle tissue and orbital fat. The muscle fibers when seen on long section showed loss of striation and in places had a teased out appearance. The fibers on cross section showed an incompletely filled muscle sheath and pale-staining amorphous substance instead of the usual clearcut muscle fibrillae. In places there appeared to be a proliferation of the perimysium. In several places there was a slight perivascular infiltration." The diagnosis was Zenker's waxy degeneration of the muscle.

Briefly, in these 4 cases there were the same clinical, operative and pathologic observations. Clinically there were exophthalmos, limitation of upward motility with diplopia and a positive result of the traction test. At operation in each case, the inferior rectus muscle was seen to be enlarged, firm and fibrotic and when removed cut with a gritty feeling. Histologically, in all 4 cases there were degeneration of muscle fibers, fibrosis and lymphocytic infiltration.

COMMENT

Etiology.—The cause of chronic orbital myositis is unknown. Tuberculosis and syphilis have often been mentioned, even though the clinical and microscopic observations have repeatedly contradicted this claim. Some investigators have ascribed it to foci of infection, but this theory has never been proved. The fact that the inferior rectus and inferior oblique muscles are so often affected naturally tends to incriminate the antrum.

Nothing in the past histories of our patients gives a clue to the cause of the condition, for none had recently been ill. None had ever had thyroid disease, typhoid or syphilis. Sex and age may be a factor, since 3 males to 1 female is the usual sex incidence and the average age is over 40. There seems to be no relationship between the age of the patient and the severity of the condition, since some of the most rapidly developing myositis reported in the literature has occurred in patients over as well as in patients under 50. Colley [24] reported a case of chronic orbital myositis in a child aged 12, in which exenteration of one orbit was followed by development of the same condition in the other orbit one year later.

Briefly, there is no positive evidence that poisoning, avitaminosis, climate, heredity, diet, allergy, glandular dysfunction, infection, trauma or occupation plays any part in the causation of this condition.

Of the various etiologic theories advanced three are worthy of note, namely, the infectious theory of Offret, the lactic acid theory of Wells and the endocrine theory of Marine, Freigold, Smelzer, Aird and others.

From microscopic studies, Offret argued that the initial lesion is subacute or chronic phlebitis of the small veins or capillaries of the orbit and that all the clinical and pathologic observations of chronic orbital myositis can be explained on the basis of this conception.

In explaining the causation of Zenker's waxy degeneration in striated muscle, Wells [33] was able to show experimentally in vivo and in vitro that the histologic changes could be produced solely by an accumulation of lactic acid in the muscles. He stated the belief that some toxin produces an excessive amount of lactic acid in the muscles and that this is directly responsible for the degeneration of the muscles. Any interference with the venous circulation, such as that hypothesized by Offret, would cause an accumulation of lactic acid, thus accentuating the degenerative process.

Experimentally it has been shown that after the injection of a thyrotropic extract made from the pituitary gland the extraocular muscles of thyroidectomized guinea pigs become enlarged and present evidence of degeneration and lymphocytic infiltration similar to that noted in chronic orbital myositis. This experimental work has been accepted in some places as an explanation for the cause of so-called malignant exophthalmos associated with thyroid disease, but so far as we know it has not been advanced to explain the cause of chronic orbital myositis.

With these theories accepted as working hypotheses, it is still necessary to explain what causes the endophlebitis of Offret's theory, to demonstrate the nature and origin of the toxin of Well's theory or to prove the presence of glandular dysfunction in chronic orbital myositis.

Gross and Microscopic Changes.—At the time of operation, most observers have noted an enlargement of the extraocular muscles, sometimes to four or five times their normal size. Grossly the muscles are described as being pale, firm, smooth, friable and cartilaginous. The belly of each muscle affected usually shows a fusiform enlargement, with the ends not much affected. Sometimes the sheaths of the muscles are loosely attached to the orbital fat and the surrounding orbital tissues. One or more of the extraocular muscles may be affected, but it is a curious fact that the inferior rectus and inferior oblique muscles have been affected more often than any of the other extraocular muscles.

A number of writers have reported cases of chronic myositis affecting only the inferior rectus or the inferior oblique muscle; others have noted enlargement only of the superior rectus or superior oblique muscle. A number of observers have reported some involvement of all of the extraocular muscles. From our experience and from what we have been able to learn from the literature, it seems that any or all of the extraocular muscles may be affected but that the muscles below the globe show a special susceptibility to this disease.

Microscopically, the histologic changes were basically the same in all our cases, consisting chiefly of fibrosis, degeneration of muscle fibers and lymphocytic infiltration. Offret, who has made a special study of the histologic changes of chronic orbital myositis, tabulated the following ones as characteristic: [34]

1. Lymphocytic infiltration throughout the orbit with a few scattered plasm cells and a tendency to follicle formation about the capillaries. This change may be seen (*a*) about the central vessels of the muscle fibers, (*b*) in the interstitial tissue, (*c*) in the muscle aponeurosis and (*d*) in and about the individual muscle fibers.

2. A fibrosis between and about the muscle fibers.

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33. Wells, H. G.: The Pathogenesis of Waxy Degeneration of Striated Muscles (Zenker's Degeneration), J. Exper. Med. **11**:1, 1909; Waxy Degeneration of Diaphragm: Factor in Causing Death in Pneumonia and in Other Conditions, Arch. Path. **4**:681 (Nov.) 1927.

34. The histologic diagnosis of chronic orbital myositis should be entrusted only to pathologists familiar with this condition, for it has been confused with a malignant process, gumma, tuberculoma, lymphoblastoma and Hodgkn's disease.

3. Marked changes in the muscle fibers themselves: i. e., edema of the stroma; lymphocytic infiltration; disappearance of the striations; vacuolation; poor staining qualities; collaginous, waxy or glassy degeneration (Zenker's [35]); changes in the nuclei; degeneration, and fibrosis.

4. Vascular changes confined to the smaller veins and capillaries, which show lymphocytic infiltration in and about the vessel walls with slight endothelial proliferation but no endarteritis.

Offret has studied the histologic changes characteristic of traumatic myositis, acute myositis, congenital fibrosis (Duane's syndrome, etc.) and degenerative myositis of vascular, fatty or amyloid degeneration. He maintained that these processes are different and distinct from the type of degeneration under discussion here. He stressed the point that chronic nonspecific orbital myositis must not be confused with syphilitic or tuberculous orbital myositis and cited several clinical and histologic studies of each to prove it.

Benedict [36] and others have stated the belief that the pathologic changes of chronic orbital myositis may vary according to the age of the process, the older processes showing more fibrosis and the more recent ones less fibrosis. Generally the orbital fat and sometimes the lacrimal gland show the same histologic picture noted in the extraocular muscles.

F. H. Verhoeff,[36] A. P. Stout,[36] Fred Stewart,[36] A. M. Pappenheimer,[36] J. S. McGavic,[36] Orlow [18] and others have insisted that the condition is essentially one of chronic degeneration of muscle fibers, that it is not to be confused with so-called pseudotumor or granuloma of the orbit and that it is a separate and distinct histologic entity. The fact that none of our patients presented evidence of involvement of the orbital fat would be an argument in favor of this view. However, a number of cases have been reported in which chronic inflammatory changes have been noted in the orbital fat as well as in the muscles. This fact has led some writers, notably Reese,[36] to express the belief that chronic orbital myositis is a pseudotumor which begins in the extraocular muscles and extends from there to involve the orbital fat. It is tempting to accept the view that chronic orbital myositis is a separate disease entity, distinct clinically and pathologically from so-called granuloma of the orbit, but we prefer to await further clinical and histologic studies before becoming dogmatic on this point.

In studying the pathologic changes of chronic orbital myositis, several observers have noted the great similarity between the histologic picture of this disease and that noted in malignant exophthalmos associated with exophthalmic goiter. In fact, the histologic pictures sometimes cannot be differentiated under the microscope. At the present time there is no satisfactory explanation why these two conditions should be so similar grossly, microscopically and sometimes clinically.

Subjective Symptoms.—The onset may be gradual or sudden in an otherwise healthy person. When it is gradual, there may be an interval of several months between the first symptom and the first visit to the physician. When it is sudden, this interval may be reduced to two weeks or less. In many of the cases reported

35. The name Zenker's waxy degeneration has been applied to these changes in striated muscle, after the pathologist who first described this change, in the rectus abdominus muscle, in patients who had recovered from typhoid fever (MacCallum, W. G.: Text-Book of Pathology, Philadelphia, W. B. Saunders Company, 1916, pp. 92 and 572). William Boyd (Text-Book of Pathology, Philadelphia, Lea & Febiger, 1938, p. 1019) stated that he had seen this change disappear spontaneously, thus giving histologic confirmation to the clinical observation that many patients may recover spontaneously if given a chance.

36. Personal communications to the authors.

in the literature the onset was rather sudden and alarming, simulating the clinical appearance of an acute inflammatory process of the orbit without constitutional signs. In all of our 4 cases the onset was gradual.

The three symptoms most complained of are edema of the lids, diplopia and exophthalmos.

1. Edema: The first symptom is usually a slight, sometimes transitory, painless edema of the lids. If this edema appears it may progress rapidly. In only 1 of our cases (case 3) did the patient notice edema of the lids before entering the hospital, and this began in the lower lid. Only one orbit was involved in each of our cases. This observation seems to be in disagreement with that of some investigators, notably Offret,[1] who stated that the second orbit becomes involved in 25 per cent of cases.

2. Diplopia: A second, and probably the most constant, subjective symptom is binocular diplopia. Soon after the onset, all of our patients noted this symptom. Patient 1 was wearing a patch over one eye to prevent diplopia and nausea. Patient 4 did not complain of double vision, although examination showed some limitation of ocular motility in the affected eye. These findings are in complete accord with those of other writers on this subject. Therefore it seems that diplopia may be considered to be one of the most consistent and most characteristic symptoms of chronic orbital myositis.

3. Exophthalmos: A third subjective symptom of this condition is exophthalmos. This was a fairly constant component in the triad of symptoms (edema, diplopia and exophthalmos) in our cases, being present in all but 1. In all cases the exophthalmos developed without apparent cause, and in only 1 was trauma associated with protrusion of the globe. In case 2 there was a temporary arrest, or a regression, of his exophthalmos. In some cases reported in the literature (Orlow,[18] Offret [1] and others) the progress of the exophthalmos had been so sudden and so great that exposure keratitis requiring enucleation developed. In a number of cases in the literature the two orbits were affected, either simultaneously or consecutively (Offret,[1] Orlow,[18] O'Brien,[22] Colley,[24] Benedict [4] and others), but in none of our cases were both orbits involved.

Objective Symptoms.—1. Edema: Examination of the lids in our cases gave essentially negative results except to reveal slight edema of the left lower lid in case 3. This edema was firm, painless and pale and did not pit on pressure. In none of the cases could a mass be felt in the lids or orbit.

Offret stated that most patients do not present a mass palpable through the skin. In Colley's [24] case, a mass could be felt through the upper lid; also, in Benedict's [4] 2 cases a mass was noted before operation. Several other observers have noted a palpable mass before operation.

2. Exophthalmos: The amount of exophthalmos in our cases varied from 2 to 4 mm. on the Hertel exophthalmometer. Some observers have recorded an exophthalmos as high as 12 mm. (Ellett) or 13 mm. (Offret), but most records in the literature fail to give the exact amount present.

The displacement of the globe in our cases was either straight forward or forward and downward, with little or no lateral displacement. The exophthalmos was not reducible or affected by the position of the head. At the time of examination, it was unilateral in all of our cases. No pulsation, bruit or tenderness was present. In cases 2 and 3 measurement of the exophthalmos was difficult because of the extreme downward rotation of the affected eye.

3. Limited Ocular Motility: Examination of the extraocular muscles with the eyes in the six cardinal directions of gaze revealed some limitation of ocular motility in all of our cases. Most movements of the affected eye controlled by the extraocular muscles were definitely limited. Especially was this true of elevation and upward rotation. None of our patients could rotate the affected eye upward beyond the horizontal plane. In 2 cases (2 and 3) the globe was fixed in a position of downward rotation equal to about 30 degrees of arc. In 3 a provisional clinical diagnosis of paralysis of the superior rectus muscle on the affected side had been considered. This finding, associated with exophthalmos, naturally led some clinicians to suspect a tumor behind the globe and above the optic nerve.

Most observers have noted a marked limitation of movement of the affected eye. Several have especially mentioned a weakness of elevation or a paralysis of the superior rectus muscle. Because of the frequency of limited upward motility, we believe that this finding should be stressed as perhaps the most important sign of chronic orbital myositis.

The limitation of movement in the affected eye is associated usually with strong secondary overactions of the associated muscles of the sound eye. When the affected eye is fixing and the patient directed to look slightly upward, for example, the unaffected eye shoots up under cover. Secondary overactions are noted in the other directions of gaze as well. This bizarre behavior makes it impossible to attribute the limited motility or the diplopia definitely to a paresis of any one muscle or set of muscles.

In only 1 of our cases was a partial ptosis noted. In 2 others there was a marked widening of the palpebral fissure of the affected eye when fixation and elevation of this eye were attempted. This produced a frightened, wild-eyed effect on that side.

Other Symptoms and Observations.—Pain is not a characteristic symptom, although 2 of our patients complained of a feeling of fulness or stiffness about the eyes. Sometimes when pain is present it was associated with sinusitis requiring surgical intervention or with exposure keratitis. It is interesting to note that in this condition, affecting primarily the extraocular muscles, pain on movement of the globe is conspicuously absent. In acute inflammatory conditions of the orbit (abscess, tenonitis, cellulitis), pain on ocular movement is a well known and characteristic symptom.

None of our patients complained of visual impairment, except in the form of diplopia, although some observers (Benedict,[4] Michail and Rusu,[16] Ellett [9b] and others) have reported failing vision, which sometimes develops suddenly.

Partial ptosis had been noted in 1 of our cases (case 1). Other observers (for example, Benedict [4] and Williamson-Noble [23]) have also reported ptosis. It must be stated, however, that as a general rule the function of the levator is not affected by this condition.

Perimetric field studies when done have given normal results. The pupillary reflexes, ocular media and fundi are usually normal. The fundi in all of our cases were considered normal.

Roentgen ray studies of the orbit show only a slight increase of soft tissue density. Roentgen ray studies of the gastrointestinal tract, skull or long bones have not been of diagnostic help.

According to Offret, puncture with a needle does not help in the diagnosis, and therefore we did not resort to this procedure in any of our cases. Reese, in discussing a case report by Lewis,[30w] stated that a diagnosis of chronic inflammatory

tumor had been made on the basis of aspiration biopsy. No mention was made of any change in the extraocular muscles, however.

Routine laboratory studies do not give any diagnostic help. Routine and special studies of the blood usually give values within normal limits. Occasionally a positive Wassermann reaction has been recorded in the literature, but this is unusual. The urine, sedimentation rate and basal metabolic rate have been normal. Electrocardiograms, Mantoux and Frei tests and tests for brucellosis have all given negative results.

A general physical examination usually fails to reveal anything significant. Arteriosclerosis has been reported in some cases, but this is to be expected in the age group affected. Busse and Hochheim reported a case in which the myocardium showed the same changes as the extraocular muscles. No evidence of thyroid disease was present in any of our cases.

Complications.—The complications of chronic orbital myositis are chiefly mechanical. They consist principally of permanent exophthalmos, exposure keratitis limitation of ocular motility, endophthalmitis and optic nerve atrophy.

Pathogenesis.—The cardinal signs of chronic orbital myositis can be entirely explained from a study of the microscopic changes in the orbit. The edema of the lids seen clinically is due to a chronic inflammatory process affecting the orbital tissues. The exophthalmos is due chiefly to an enlargement of the extraocular muscles, with extension perhaps of this chronic inflammatory process to the neighboring tissues. The limitation of ocular motility and the inextensibility of the affected muscles are due directly to the inflammation, fibrosis and degeneration present in the extraocular muscles themselves.

Prognosis.—The prognosis for spontaneous recovery is better if operation on the orbit is avoided. Some patients observed by us but not operated on have regained normal or almost normal ocular motility in from six months to a year. Those who have had biopsies of the extraocular muscles have almost without exception been worse for the surgical procedure and have sustained some degree of permanent limitation of ocular motility.

The exophthalmos usually has a tendency to regress spontaneously after the disease has run its course. Sometimes endophthalmos develops, though generally some degree of exophthalmos remains for years.

So far as sight is concerned, the prognosis is governed mostly by how much damage has occurred to the cornea from exposure. Most patients retain normal or almost normal vision, though some have become blind from extensive corneal scars. Others have suffered from optic nerve atrophy and several have lost one or both eyes from endophthalmitis.

Diagnosis.—A positive diagnosis of chronic orbital myositis cannot be made without a careful microscopic study of the extraocular muscles. It is desirable however, to make this diagnosis whenever possible without resorting to surgical intervention, since it has been shown that an operation cannot be performed with impunity. Several observers have reported an acute exacerbation of the exophthalmos with loss of the eye following a biopsy, and all are agreed that operation generally makes the condition worse. Therefore it is most essential in making the diagnosis that one keep the clinical features of the disease clearly in mind. A history of thyroid disease or of thyroidectomy makes the diagnosis much easier.

It is evident from the negative results of roentgen ray, laboratory and physical examinations of patients with chronic orbital myositis that the clinical diagnosis of this condition must depend on the diagnostic acumen of the ophthalmologist. He

must rely almost entirely on the clinical signs, the chief of which are edema of the lids, exophthalmos and diplopia. He must rule out all other orbital conditions producing this triad of symptoms, including the infectious processes of the orbit, such as periostitis, tenonitis, cellulitis, thrombosis of the cavernous sinus and actinomycosis. Also, he must exclude the noninflammatory diseases, such as malignant or benign tumor of the orbit, neoplastic disease of the hemopoietic system, mucocele, hematoma, arteriovenous aneurysm, Schüller-Christian disease with exophthalmos, metastatic lesions, paralysis of the extraocular muscles, axial myopia and certain systemic diseases (e. g., scurvy, rickets, chloroma and trichinosis).

In most of the conditions enumerated, the correct diagnosis usually presents no difficulty. In some cases of exophthalmos, however, to differentiate between some other condition and chronic orbital myositis by means of the clinical signs alone may be impossible. The number of exenterations done because of a suspected malignant process is mute testimony to this fact.

Traction Test: Because of the diagnostic difficulty, we have utilized a simple test which has proved to be of some diagnostic value in differentiating the exophthalmos of chronic orbital myositis from exophthalmos due to other causes. The test consists simply of determining the passive movements of the globe in the fields of limited ocular movement. This is done by first cocainizing the conjunctiva and episcleral tissues with 10 per cent cocaine hydrochloride (4 per cent cocaine or 0.5 per cent pontocaine hydrochloride is less effective) and then applying a fixation forceps to the tendon of the extraocular muscle in question and exerting traction in the field of limited ocular motility. For example, if there is an apparent paralysis of the superior rectus muscle with limited elevation, a wisp of cotton saturated with 10 per cent cocaine hydrochloride is placed in contact with the episcleral tissues over the tendon of the inferior rectus muscle for a few minutes. A fixation forceps is then applied to the tendon and traction exerted upward in the field of action of the superior rectus muscle. If the poor elevation of the globe is due to paresis of the superior rectus muscle, no difficulty will be encountered in elevating the globe. If, however, the inferior rectus muscle is inelastic and fibrous, as it often is in chronic orbital myositis, then the globe will remain stationary even though strong traction is exerted upward. This simple test when the result is positive therefore rules out paralysis and discloses the restrictive nature of the limitation of the ocular motility.

We found this test to give positive results in all 4 of our cases and subsequently showed histologically the presence of fibrosis of one or more of the extraocular muscles in each case.

In case 1 the test was first carried out with the patient under ether anesthesia during an exploratory operation. The patient presented moderate exophthalmos, downward displacement of the globe and clinical signs of paralysis of elevation. At the time of operation it was clearly evident that no mass was present above the globe to produce the exophthalmos and the downward rotation of the globe. A muscle hook inserted under the tendon of the inferior rectus muscle with traction upward showed that this muscle was inextensible and that the inextensibility was solely responsible for the so-called "paralysis of elevation."

In 2 other cases (2 and 3) the traction test gave positive results preoperatively. At operation in each case the inferior rectus muscle was found to be grossly inelastic, firm and fibrotic. In the fourth case the inelasticity of this muscle was demonstrated at operation.

This simple test has been helpful in differentiating clinically the limitation of ocular motility associated with chronic orbital myositis from that due to other

causes. No claim is made for its originality or infallibility, since it is obvious that it would give a positive result in a case of congenital fibrosis of the external rectus muscle (Duane's syndrome). Also, it might be misleading when traumatic or inflammatory adhesions between the extraocular muscles and the walls of the orbit were present. That such a situation can occur was demonstrated in a case of mixed tumor of the lacrimal gland seen by one of us (J. H. D.), in which the external rectus muscle was affected, producing definite limitation of ocular movement.

Having in mind the clinical picture of chronic orbital myositis and the limitations of the traction test, we would suggest the following clinical signs as characteristic of this disease: 1. Painless edema of lids with gradual or sudden onset. 2. Moderate to severe exophthalmos. 3. Limitation of ocular movement, especially upward. 4. A positive result of the traction test.

Treatment.—There is no recognized form of treatment for chronic orbital myositis. Surgical intervention should be avoided if possible and if carried out should be limited to the treatment of the complications. A biopsy should not be done unless the diagnosis cannot be made in any other way. Most observers are agreed that operation on the orbit or on the muscles themselves often exaggerates the exophthalmos and generally makes a spontaneous recovery with satisfactory ocular motility impossible.

Should the integrity of the cornea become threatened by exposure, intermarginal sutures should be inserted or adhesions formed. If the conjunctiva prolapses an attempt should be made to replace it or to keep it covered with petrolatum. An evisceration or an enucleation may have to be done for complicating endophthalmitis.

Roentgen ray therapy to the orbit has been tried with indifferent results. According to Niall,[13d] such treatment to the pituitary gland has had no effect on this disease. This form of therapy is based on the theory that the thyrotropic hormone or some unknown secretion from an overactive pituitary gland may be the cause of the exophthalmos. Antisyphilitic treatment with the iodides and the arsenic preparations has not been of proved value, nor has it prevented extension of the disease to the opposite side. Thyroid extract has either had no effect or has exaggerated the exophthalmos. Intravenous administration of hypertonic solutions has been of questionable value. Cervical sympathectomy has been of no help.

SUMMARY

Chronic orbital myositis is a nonspecific, chronic inflammatory disease of the extraocular muscles of unknown causation. It is characterized histologically by lymphocytic infiltration, fibrosis and degeneration of muscle fibers. Grossly the muscles are enlarged, pale and cartilaginous, and they cut with a gritty feeling. Clinically the disease is characterized by (1) painless edema of the lids of gradual or sudden onset; (2) moderate to severe exophthalmos; (3) limitation of ocular movement, especially upward, and (4) inelasticity of the affected muscles as shown by the traction test described.

CONCLUSION

A positive diagnosis of chronic orbital myositis can be made only by biopsy of the affected muscles. This is to be avoided if possible, however, because of the untoward results attending surgical intervention. The correct diagnosis can generally be made from the clinical signs listed. The prognosis for spontaneous recovery is good if surgical intervention can be avoided. Treatment should be conservative and directed primarily toward preserving the integrity of the cornea.

This is best accomplished by such means as the instillation of liquid petrolatum to prevent drying of the cornea, petrolatum dressings, insertion of intermarginal sutures or formation of adhesions between the margins of the lids.

Dr. Ludwig von Sallmann translated much of the foreign literature listed in the footnotes.

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DISCUSSION

DR. JOHN S. MCGAVIC (by invitation): Dr. Dunnington and Dr. Berke have reported a group of cases representing a distinct clinical entity not previously reported. Orbital myositis has been described as a complication of thyroid disorders, as a condition secondary to adjacent inflammation (abscess, cellulitis and scleritis) in trichinosis and as an isolated histologic change not associated with any characteristic clinical picture.

It has been my privilege to study the biopsy material in their cases. Only two histologic entities are likely to be confused with chronic orbital myositis: (1) granuloma and (2) myositis of hyperthyroidism.

Birch-Hirschfeld first and later Benedict and Knight described what they chose to call pseudotumor of the orbit. This term was meant to indicate how closely this lesion resembles true tumor of the orbit, many patients having had exenteration of the orbit after a diagnosis of neoplasm. The lesion they described is a granuloma. Unfortunately the term pseudotumor remained and has come to be thought of as the ophthalmologist's name for granuloma of the orbit.

If one is to use this odd name pseudotumor, one should use it as a heading under which all non-neoplastic lesions of the orbit are placed. The ophthalmic pathologist will better serve the clinician and avoid confusion in his own mind if he reports "pseudotumor: granuloma," "pseudotumor: hematoma," "pseudotumor: myositis," "pseudotumor: cyst" "pseudotumor: cholesteatoma," and so on, for each of these is as much a pseudotumor as is the granuloma. In fact, pseudotumor should be only a clinical term.

Little will be accomplished in clearing the confusion regarding the causes of exophthalmos until the biopsy tissue already available for histologic study is classified more specifically.

The first section to be shown is from granuloma of the orbit. Histologically one finds tubercles made up of giant cells, some endothelial cells, lymphocytes, plasma cells and a few polymorphonuclear cells associated with a large amount of highly vascularized fibrous connective tissue. The blood vessels show intimal proliferation. Tuberculosis and syphilis can be differentiated readily. Muscle tissue may be involved along with the orbital tissues, but there is no apparent selective affinity for muscle tissue. Many such tumors have been studied at biopsy, and, although muscle tissue may be found to be involved, one does not read in the operative note that a large portion of one rectus muscle had to be removed to allow excision of the mass.

Dr. Berke stated that in chronic orbital myositis the muscle itself is so intimately involved that resection of the bulk of it may be necessary and in fact was in several of the cases reported.

When one looks at the sections, one finds much more muscle tissue than is usual in biopsy specimens from granulomas. There are focal or diffuse infiltrations of lymphocytes and plasma cells, some fibrosis and various amounts of degeneration of muscle fibers with loss of cross striations. The last-mentioned change is not usual in granulomas. Whether or not degeneration of muscle fibers is primary or secondary I do not know, nor has this information been forthcoming from the general pathologists. It seemed to me that there should be no difficulty in distinguishing granuloma from chronic orbital myositis histologically. To check my opinion, I took a group sections from what ophthalmologists call granulomas mixed

with the sections from muscles affected with myositis to a general pathologist who has done a good deal of work with muscle tissue, and he separated the sections from granulomas into a group by themselves, although he had not known that ophthalmologists have a granuloma peculiar to their specialty.

After sections representing chronic orbital myositis were compared with biopsy material from patients with malignant exophthalmos associated with hyperthyroidism and hypothyroidism, it was found that there was not sufficient difference in the histopathologic changes to differentiate the two conditions. However, specimens from patients with thyroid disease showed marked variation from one muscle to another, and the material often was not adequate for proper study. It is possible that further clinical investigation will show that some of these specimens actually did not represent thyroid disease. It may be found that the cause of the two conditions is fundamentally the same, with the thyroid dysfunction a contributing factor rather than a directly causative factor even when the patient has true thyroid disturbance. At any rate in the cases reported there was more than adequate clinical evidence of the absence of thyroid disease.

Dr. Dunnington and Dr. Berke not only have presented a new and distinct clinical entity and described a helpful diagnostic sign, but have done ophthalmologists a service by emphasizing that they must classify the so-called pseudotumors according to their specific histologic types as far as this is possible.

DR. JOHN H. DUNNINGTON: In presenting this subject we have considered the differences of opinion that may exist with regard to the entire clinical picture, but we feel that the condition is a distinct clinical entity. Some so-called cases of pseudotumor undoubtedly are instances of chronic orbital myositis, but the pathologic changes in most of these cases are very different from that found in our cases. The term pseudotumor in my opinion is a poor one and should be relegated to the scrap heap. I should like to have the pathologists more specific in their diagnoses. I believe that if they really made an effort they could subdivide the cases into their proper groups.

There are a few points which I wish to reiterate:

1. The apparent paralysis of elevation produced by the inelasticity of the inflamed and fibrotic inferior rectus muscle. Limitation of elevation is such a constant finding in this disease that the presence of the disease should always be suspected in a case of unilateral exophthalmos with impaired upward motility of the eye. Other conditions can produce such a mechanical interference with elevation, e. g., traumatic enophthalmos with fracture of the floor of the orbit. The traction test should always be employed to prove the cause of limited motility, and we have found it to be of great value. It is interesting that extreme traction on the inelastic muscles produces little or no discomfort.

2. The similarity between the pathologic changes in the affected muscles and those observed in malignant exophthalmos. This makes one wonder whether or not in some instances the two conditions have not been confused. In a few of the reported cases of malignant exophthalmos the basal metabolism was more or less normal prior to thyroidectomy, and in many diplopia was an early complaint. The frequent spontaneous recession of the exophthalmos in the two conditions is another striking similarity. Future investigation may prove that orbital myositis is the primary lesion in malignant exophthalmos and becomes aggravated by thyroidectomy. Diplopia in associaton with any slight exophthalmos should make one suspicious of the orbital nature of the lesion.

3. The prognosis is much better if operation is avoided. A biopsy is essential for accurate diagnosis but retards recovery and produces further cicatrication in the orbit.

VITAMIN E (WHEAT GERM OIL) IN THE TREATMENT OF INTERSTITIAL KERATITIS

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Interstitial keratitis remains one of the most frequently encountered complications of late congenital syphilis. From one third to one half of patients with congenital syphilis have this disorder at some period of their lives.[1] The fact that it is most resistant to antisyphilitic therapy as compared with other lesions[2] has brought forth the suggestion that other factors besides spirochetal invasion of the cornea are responsible. Trauma,[3] allergic states[4] and endocrine disturbances,[5] the last in view of the frequent onset of symptoms at puberty, have all been suggested as contributory causes. Sandler's[6] and Arena's[7] reports on rapid clearing of symptoms in cases of recent interstitial keratitis in from four days to three weeks after administration of sulfonamide compounds suggests that in some cases the probable cause of the acute flare-up is an organism or virus responsive to such therapy. No reports are as yet available, however, on results of this treatment in cases of long-standing interstitial keratitis. The favorable results of riboflavin therapy reported in several cases by Kruse, Sydenstricker, Sebrell and Cleckley[8] and by Sydenstricker, Kelly and Weaver[9] have brought forth the suggestion of dietary deficiency as a contributing cause.

The accepted method of treatment in the past consisted of at least two years of injections of compounds of arsenic and bismuth combined with large doses of iodides.[10] Klauder and Vandoren[11] in a recent analysis of results of treatment of 532 patients found that the best results were obtained when artificial fever was

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From the Neurological and Artificial Fever Therapy Services, Elliot Hospital; the Syphilis Therapy Service of the New Hampshire State Hospital and the New Hampshire State Department of Venereal Disease Control.

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combined with routine treatment. While the incidence of relapses with other methods ranged from 13 to 18 per cent, it was only 2 per cent when routine treatment was combined with fever therapy. The same authors found iodides of no value in interstitial keratitis.

PRESENT STUDY

The beneficial effect of vitamin E (wheat germ oil) on the rate of absorption of tissue exudates as observed in cases of arthritis deformans,[12] in cases of congenital nonobstructive hydrocephalus [13] and in selected cases of pseudohypertrophic muscular dystrophy [14] has prompted the use of vitamin E in the treatment of resistant interstitial keratitis. The action of the vitamin E was considered to be nonspecific, although the possibility of dietary deficiency as a contributing cause in the development of the keratitis was given consideration in view of the poor nutritional state of many of the patients. The patients treated were seen through the venereal disease control clinics of the state of New Hampshire and the syphilis therapy service of the New Hampshire State Hospital. In the past patients in the clinics with interstitial keratitis who did not respond to routine arsenic-bismuth therapy combined with oral administration of vitamin B complex were treated with artificial fever. While the fever almost always brought relief from photophobia and corneal congestion and hastened absorption of exudates of recent origin, its effect on corneal opacities of long standing was limited. The first 2 patients treated with vitamin E received artificial fever concurrently, although administration of the vitamin was begun before the artificial fever treatments were and was continued during the period of the treatments and for a number of months after they were completed. Since then, however, it has not been found necessary to administer artificial fever to any of the treatment-resistant patients in view of the improvement observed with vitamin therapy alone. Also, in 4 cases in which artificial fever treatments had been carried out months earlier for neurosyphilis and associated interstitial keratitis with little absorption of corneal opacities as a result, vitamin E therapy was started a number of months after the completion of the fever therapy, when no further improvement from the latter was expected.

PATIENTS TREATED

This study began in March 1940, when treatment of the first patient with vitamin E was begun. A total of 10 patients with advanced interstitial keratitis have been treated to date. Nine of the patients were female, and 1 was male. The oldest patient was 42 and the youngest 7 years old. The duration of the ocular symptoms was thirty-five years in the oldest patient and three months in the youngest. All of the patients had previously received one or more courses of arsenic-bismuth therapy except 1, who was treated for two years with mercury inunctions and acetarsone administered orally. One patient received intramuscular injections of foreign proteins. Two of the patients received artificial fever therapy concurrently with the vitamin therapy, and 4 received artificial fever therapy from four months to one year before having been given vitamin E for associated symptoms of

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involvement of the central nervous system. Four patients received vitamin E therapy but no artificial fever. Antisyphilitic therapy was administered only to patients who had not received adequate treatment in the past. None of the patients in this group received injections of arsenicals or bismuth preparations while receiving vitamin E.

All patients had severe limitation of vision in one or both eyes. Four patients had only perception of light in one eye, and 2 of the 4 had perception of fingers in the better eye with extensive dense opacities and scars over both corneas. Another patient had an opacity involving the entire cornea of one eye without perception of light in that eye but with normal vision in the other eye. She had had a partial tattooing of the involved cornea performed for cosmetic reasons. In the others the vision in the better eye varied from perception of fingers to blurring of vision. Photophobia, vascular congestion and superficial and deep corneal opacities were present in all cases.

METHOD OF TREATMENT

The vitamin E was administered at first in the form of a mixture of wheat germ oil (Lilly) with vitamin B complex [15] (Lederle), 1 part of the oil to 3 parts of vitamin B complex, in doses of 8 to 12 cc. daily. The vitamin B complex was added in view of its previously observed synergistic action with vitamin E. With the introduction of wheat germ oil concentrates, 50 mg. mixed tocopherol capsules (Lilly) or (Winthrop) were given instead, one capsule once or twice daily, usually in combination with one or two capsules of vitamin B complex. With improvement in the patient's condition this dose was gradually reduced, so that in the end a maintenance dose of 50 to 100 mg. of mixed tocopherols weekly appeared ample. Several patients received this combined vitamin therapy for twelve to eighteen months, showing gradual but continuous absorption of corneal opacities and scars. All patients except 1 who received artificial fever treatments had involvement of the central nervous system and had previously been given an average of eight to ten inductothermy fever treatments with at least two hours of fever at a temperature over 104 F. at each treatment. Two began to receive vitamin E before the fever treatments were started, while with the other 4 patients the vitamin E therapy was not started till a number of months had elapsed after the fever treatments had been completed.

Three of the 4 patients who received vitamin therapy alone had received continuous antisyphilitic therapy previously for at least two years. The keratitis in 3 was of long standing, and in 2 the flare-up developed during antisyphilitic therapy. Two patients in this group received 1 to 2 mg. of riboflavin three times daily for several months before vitamin E therapy was begun, without any effect on the corneal exudates, although the photophobia and the circumcorneal congestion were somewhat relieved. One patient received no antisyphilitic therapy before the vitamin therapy except oral administration of acetarsone for two years and mercury inunctions four years earlier.

RESULTS

The 10 patients treated can be divided into three groups: 2 with advanced keratitis who received vitamin E therapy shortly before they were given artificial fever treatments, 4 who received artificial fever therapy four to twelve months before being given vitamin E because of failure of the cornea to clear after artificial fever and 4 who received only vitamin E therapy. All showed changes for the

15. Each 4 cc. of the vitamin B complex contained thiamine hydrochloride 1.5 mg., riboflavin 0.8 mg., nicotinamide 10 mg., pyridoxine hydrochloride 0.13 mg. and pantothenic acid 0.2 mg.

better within a week after vitamin therapy was begun. Disappearance of photophobia and corneal congestion were the first changes noted. These were followed by obliteration of the capillary loops at the corneoscleral junction and wherever they invaded the cornea. The disappearance of pathologic vascularization and clearing of superficial exudates and opacities usually took place at the end of two or three weeks. The absorption of deep opacities was much slower, and in 1 of the most severely affected patients the cornea did not clear entirely for eighteen months after treatment was started.

Of the first 2 patients who received artificial fever treatments concurrently with vitamin E therapy, 1 had only perception of light in one eye and perception of fingers at a few feet in the better eye after eight months of intensive antisyphilitic therapy, including injections of foreign protein. This patient was given vitamin therapy for a week before she was treated with artificial fever, and considerable relief from the photophobia and circumcorneal injection and some clearing of superficial opacities occurred before she received the first fever treatment. Complete disappearance of photophobia and vascular proliferation was observed after ten fever treatments, at the end of one month. The vision in the right eye was still, however, only 20/200 and that in the left eye 20/80. While the iris could be seen clearly at this time because of absorption of the superficial exudates, the fundus in the more severely affected eye could not be visualized because of residual deep opacities. Absorption of the opacities continued gradually, and six months later the vision in the better eye was 20/20, with complete disappearance of all opacities and salmon patches. The fundus of the more severely affected eye could be seen for the first time. Eighteen months after treatment was begun, complete absorption of the deep opacities and scars had taken place and the visual acuity was 20/20 in each eye. This patient continued with vitamin E in gradually decreasing doses all during this period.

A similar absorption of deep opacities took place in the second patient, although the visual impairment was less severe and the opacities less marked.

In the patients who received artificial fever therapy months before the vitamin therapy, the absorption of deep corneal opacities became apparent only after the administration of vitamin E. Again the absorption of the opacities was gradual but continuous, with a constant gain in visual acuity. One patient in this group had had her cornea partly tattooed for cosmetic reasons, as she had completely lost her vision in that eye as a result of keratitis in childhood, and return of perception of light and fingers took place after the nontattooed part of the scar had gradually become absorbed. The artificial fever was administered to this patient primarily for associated dementia paralytica. It is possible that the artificial fever administered several months earlier had played a part in initiating the absorption of the thick scar tissue.

In the patients who received vitamin therapy alone, the first evidence of improvement was manifested in disappearance of photophobia and corneal congestion, this usually within a week or ten days. In 2 patients in whom the opacities were less severe, complete clearing of the cornea took place within a month. In a patient who had had the condition for several years and had failed to respond to continuous antisyphilitic therapy, with only perception of light in the better eye owing to deep and superficial corneal opacities and advanced vascularization of the cornea, gradual absorption of the opacities took place and after twelve months of vitamin E therapy normal vision resulted, with complete clearing of both corneas.

In the fourth patient in this group, who had only doubtful perception of light in the affected eye associated with photophobia, capillary proliferation and obscura-

tion of the iris by thick exudate, the photophobia had disappeared at the end of two weeks, with sufficient absorption of exudate so that the iris could be seen clearly. Perception of fingers at a distance of 10 feet (300 cm.) returned twenty-four days after therapy was begun. The optic fundus could be seen clearly at this time. At the end of one month complete clearing of the cornea had taken place. It is significant that this patient had received no antisyphilitic therapy for at least four years before the onset of ocular symptoms and previously had received only acetarsone orally and mercury inunctions for two years. No antisyphilitic therapy had been administered to this patient since the onset of ocular symptoms, and none of the other patients in this group received antisyphilitic therapy while receiving vitamin E.

Two of the patients in this group received riboflavin in doses of 3 to 6 mg. daily for several months before they were given vitamin E. As observed in patients in this group and others the effect of the riboflavin was mainly to relieve the photophobia and capillary congestion; it did not appear to hasten the absorption of corneal exudates. It is possible, however, that the riboflavin content of the vitamin B complex which these patients received—about 1.6 to 2.4 mg. daily—was of some therapeutic value in relieving the photophobia and corneal congestion. The vitamin B complex was administered primarily, however, for its previously observed synergistic effect on vitamin E, and absorption of exudates continued when its use was temporarily discontinued.

REPORT OF CASES

CASE 1.—I. A., a 10 year old girl, was admitted to the Elliot Hospital on March 13, 1940 for treatment of resistant interstitial keratitis. The symptoms had begun about one year before admission, and although she was immediately given arsenic and bismuth therapy, no improvement resulted. Ophthalmologic examination on February 27 revealed considerable photophobia of both eyes, increased vascularization of both corneas and limitation of vision to 20/200 in the right eye and 20/100 in the left with and without glasses. On admission she showed slight notching of the teeth, prominence of the frontal bosses, photophobia, bilateral corneal opacities, nystagmoid movements of both eyes and saber shins. The vibration sense was reduced to one half over the tibias. The biceps and triceps reflexes and the knee and ankle jerks were not elicited. Examination of the spinal fluid disclosed normal pressure and dynamics, 5 lymphocytes, a total protein content of 25 mg., and a colloidal gold curve of $0^{1}0$. The Hinton reaction of the blood was positive and that of the spinal fluid negative.

The patient was given wheat germ oil and vitamin B complex in a 1:6 mixture, 4 cc. three times daily, and was given nine artificial fever treatments at three day intervals, with the temperature above 105 F. for about two hours at each treatment. On March 26 the photophobia had disappeared, the patient was able to dispense with dark glasses and the corneal opacities were almost entirely absorbed. She was discharged on April 20 with the vision greatly improved, so that she was able to return to school.

On Feb. 25, 1942 studies of the spinal fluid gave negative results, as did the Hinton test of the blood. The vision appeared good without glasses, although nystagmoid movements of both eyes persisted. Both corneas appeared clear and showed no evidence of nebulas or opacities.

CASE 2.—D. S., a 9 year old girl, was admitted to the Elliot Hospital on June 11, 1941, for treatment of resistant interstitial keratitis. Symptoms of visual failure in the right eye had developed gradually about a year before. As the patient failed to improve with routine antisyphilitic therapy, she was admitted to a hospital five months later for foreign protein therapy while continuing with arsenic-bismuth therapy. While she was receiving this treatment the left eye became involved.

Examination of eyes on admission showed marked photophobia; both corneas were steamy, with a ground glass appearance. The iris could not be seen, extensive vascularization of the cornea and injection of the sclera were present bilaterally, vision was limited in the right eye to perception of light and in the left eye to perception of fingers at about 2 feet (60 cm.). The fundus could not be visualized in either eye. The patient was given wheat germ oil and vitamin B complex in a mixture of 1:5, 4 cc. three times daily, and one capsule of mixed tocopherols daily.

On June 18 the cornea appeared less steamy and the photophobia was much less pronounced. On June 20 she was able to perceive fingers with her right eye at a distance of about 1 foot (30 cm.).

On June 30, after two fever treatments and continuation of vitamin therapy, she was able to perceive fingers with the right eye and the four of spades at 12 feet (365 cm.) with the left eye. On July 8 she was able to see small objects with the right eye, and the photophobia had completely disappeared. The fundus still could not be seen satisfactorily in either eye. On July 28 she had completed ten artificial fever treatments while continuing with mixed tocopherols, one capsule daily. The corneal exudate was greatly reduced, so that the iris in each eye could be clearly seen.

On August 28 the superficial exudates and opacities had disappeared entirely from the left eye and the vascular congestion and capillary proliferation had become obliterated.

On September 3 an ophthalmologic recheck showed the vision to be 20/20 in the right eye and 20/80 in the left. The right cornea showed a considerable number of interstitial opacities. There were many precipitations in the posterior surface of the left cornea. In the left eye the anterior surface of the cornea, the anterior chamber and the lens were clear. The right fundus could not be visualized, but the left could be seen clearly.

When the eyes were rechecked on November 4 the vision was 20/100 in the right, 20/50 in the left and 20/40 with glasses. The patient continued with the wheat germ oil and vitamin B complex in a mixture of 1:3, 4 cc. daily, and took 50 mg. of mixed tocopherols weekly. The opacities disappeared entirely from the left eye but persisted in the right.

On March 3, 1943 the patient had 20/20 vision in each eye. All the deep exudates had disappeared from the right eye, the fundus could be clearly seen and there were no residuals except a pinpoint area at the lower part of right cornea, the remainder of a thick scar which previously had covered the entire lower part of the cornea and gradually had become absorbed. She has continued to take 50 mg. of mixed tocopherols once weekly, and it is expected that the pinpoint scar will eventually disappear.

CASE 3.—E. D., a woman aged 24, was seen on Aug. 27, 1940, at the Manchester, N. H., venereal disease clinic. She had had interstitial keratitis for several years, and although she had in the past received fifty-one injections of an arsenical and 107 of a bismuth preparation, the condition had progressed. When examined she showed only perception of light in the left eye, and there was complete obscuration of the left cornea by thick exudate, so that the iris could not be seen. The right cornea showed superficial and deep opacities. There was severe photophobia associated with increased vascularization. She was given 1 mg. of riboflavin three times daily, vitamin B complex orally and instillations of atropine sulfate in both eyes. Except for some improvement in the photophobia and possible reduction in extent of the vascularization, the riboflavin had no effect on the corneal exudates. The patient still required dark glasses because of photophobia.

On May 2, 1941, therapy with mixed tocopherols, 50 mg. daily, and vitamin B complex, one capsule daily, was begun. Two weeks later there was considerable clearing of the exudates in the left eye, and she was able to dispense with dark glasses except in bright sunlight. The dose was raised to 100 mg. of mixed tocopherols daily. By August 28 the opacities had disappeared from the right eye and could be seen in the left cornea only with oblique illumination. The patient has continued to receive 100 mg. of mixed tocopherols daily and one capsule of vitamin B complex. On October 27 both fundi could be seen clearly for the first time.

On Feb. 8, 1942, complete clearing of both corneas was noted. The patient has been able to return to a gainful occupation. For the last month she has been receiving wheat germ oil and vitamin B complex in a 1:5 mixture, 4 cc. daily, as a maintenance dose.

CASE 4.—K. W., a 14 year old girl, was seen through the venereal disease clinics of New Hampshire on Oct. 3, 1942 because of a severe relapse of interstitial keratitis of two months' duration. She had received intravenous and intramuscular therapy for two years for an attack of interstitial keratitis. The attack lasted about a year, gradually clearing after leaving many superficial and deep corneal opacities. The examination showed severe photophobia, circumcorneal congestion and capillary dilatation and bilateral corneal opacities because of which the disks could not be seen clearly. She was given wheat germ oil and vitamin B complex in a 1:3 mixture, 4 cc. three times daily. In one week the photophobia had disappeared, and at the end of a month the corneal opacities had become entirely absorbed. She received no antisyphilitic therapy during this recurrence of symptoms, and the improvement resulted from the vitamin therapy alone.

COMMENT

The gradual and continuous absorption of opacities of long standing in patients with interstitial keratitis receiving vitamin E therapy, irrespective of the amount of antisyphilitic therapy they have received, suggests the possibility that other factors besides spirochetal invasion are responsible for the corneal changes. Sydenstricker and his associates [9] have suggested dietary deficiency as an important contributing cause besides syphilis. The lack of an adequate supply of vitamins in the diet would result in a diminished resistance of epithelial membranes already weakened by syphilis against trauma, infection or the demands of puberty. Dam [16] has demonstrated that deprivation of vitamin E will produce changes in capillary permeability, associated with diffuse hemorrhages and exudation of plasma, in chicks. While this would suggest that a similar causation is responsible for disturbances in capillary permeability in human beings which have in the past responded to vitamin E therapy, there is also the probability that the beneficial results from administration of the vitamin are due to the nonspecific effect of the vitamins on selected tissues in the body, irrespective of whether or not the "biochemical lesions" were caused by deprivation of vitamins. Dam [16] has also reported reversal to normal in capillary permeability in vitamin E–deficient chicks after administration of lipocaic or reduction in the concentration of soluble salts in the diet while vitamin E was withheld. This nonspecific effect of other therapeutic agents in vitamin deficiency states would make it appear probable that vitamins in a like manner are capable of producing beneficial effects in certain pathologic conditions, not because of replacement of a deficiency, but because the cellular lesions in these conditions are similar to those produced by vitamin deprivation although they are of dissimilar causation. This would explain the analogous effect of vitamin E on the rate of absorption of tissue exudates in cases of interstitial keratitis and of unrelated pathologic states having increased capillary permeability as their common denominator.

Vitamin E as found in wheat germ oil and wheat germ oil concentrates, like vitamin B complex, probably contains several factors, each effective in its own sphere. Besides its effect on capillary permeability and its neurotropic and myotropic factors,[17] vitamin E has been found in the past to prevent excessive proliferation of fibrous tissue in muscles [18] and joints,[12] as well as in the central nervous system.[19] It is probable that its administration, besides increasing the rate of absorption of corneal opacities, prevents excessive formation of scar tissue. Although the vitamin B complex was given simultaneously with the vitamin E primarily for its previously observed synergistic action with the latter,[14] there is no doubt that the 1.6 to 2.4 mg. of riboflavin thus received daily helped to relieve some of the photophobia and circumcorneal congestion. Its effect on the rate of absorption of corneal opacities could be discounted in view of its failure to produce such improvement when administered in conjunction with artificial fever therapy or routine treatment. Also, riboflavin when administered in doses of 3 to 6 mg. daily to 2 patients several months before they had been given vitamin E

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17. Einarson, L., and Ringsted, A.: Effect of Chronic Vitamin E Deficiency on the Nervous System and Skeletal Musculature in Adult Rats: A Neurotropic Factor in Wheat Germ Oil, London, Oxford University Press, 1938, p. 127.

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failed to produce any perceptible absorption of corneal opacities. The accompanying photophobia and vascular proliferation were definitely improved, however. For this reason it would be advantageous to administer riboflavin in larger doses than the amount these patients received in the vitamin B complex given with the vitamin E. Johnson and Eckardt [20] have suggested that the capillary proliferation from the limbus into the cornea as observed in various forms of keratitis is the result of an attempt by the organism to overcome local asphyxia in the avascular tissues of the cornea by making additional oxygen available for the cells of the cornea. Riboflavin, according to these authors, through its chemical effect makes improved oxidation of the corneal cells possible, and as a result excessive vascularization becomes unnecessary. Vitamin E was observed to produce similar results by reducing excessive capillary proliferation and also caused gradual obliteration of these capillaries. The exact mechanism involved remains unknown, however.

Besides the improvement in the condition of their eyes, all the patients treated showed a notable change for the better in their physical status while receiving vitamin E and vitamin B complex. A similar favorable response has been observed in patients with tabes dorsalis [21] and other forms of neurosyphilis.[22] This would suggest the possibility that patients with latent syphilis would benefit from an increased intake of vitamin E and vitamin B complex irrespective of the amount of antisyphilitic therapy they had received.

That concurrent antisyphilitic therapy is not always needed to produce improvement in interstitial keratitis is evident from the fact that 3 patients received no antisyphilitic treatment while undergoing combined vitamin therapy. One patient had had no treatment for four years before the keratitis developed and her previous treatment had consisted of oral administration of acetarsone alternating with mercury inunctions. She received no antisyphilitic therapy during the time she received the vitamin E, and clearing of the cornea resulted after four weeks of therapy with vitamin E and vitamin B complex. Her total vitamin intake during this period consisted of 900 mg. of mixed tocopherols, 30 cc. of wheat germ oil and 180 cc. of vitamin B complex, with a total intake of 35 mg. of riboflavin. This would tend to support a view entertained by some physicians that antisyphilitic therapy possibly plays a minor part in the cure of interstitial keratitis although it is of much value in preventing relapses and minimizing some of the after-effects. The safest procedure would be, therefore, to combine administration of vitamin E and vitamin B complex with antisyphilitic therapy if the patient is considered to have received inadequate treatment in the past and to continue with the vitamin therapy till all opacities have disappeared, with the dose gaged according to the patient's needs. If needed, artificial fever therapy should be used in conjunction with administration of the vitamins and chemotherapy in the treatment of patients with resistant keratitis.

SUMMARY AND CONCLUSIONS

Ten patients with interstitial keratitis were treated with vitamin E (wheat germ oil and wheat germ oil concentrate). All had received ample antisyphilitic therapy in the past. Four had received artificial fever therapy a number of months earlier because of associated involvement of the central nervous system without

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21. Stone, S.: Vitamin B and E Therapy in Tabes Dorsalis, J. Nerv. & Ment. Dis., **95**:156, 1942.

22. Stone, S.: Non-Specific Therapy of Neurosyphilis: Results from Intraspinal Administration of Thiamin Chloride Combined with Vitamin B Complex and Vitamin E (Wheat Germ Oil), Urol. & Cutan. Rev. **66**:714, 1942.

its markedly affecting the course of the keratitis. Two patients were given artificial fever treatments shortly after therapy with vitamin E was begun.

Vitamin E was mainly effective in hastening absorption of superficial and deep corneal exudates; it helped to relieve the associated photophobia and reduce excessive corneal vascularization and circumcorneal congestion. In cases of long-standing involvement with extensive opacities and corneal scarring, its administration for a period of months has produced a gradual and continuous clearing of the cornea with a return of normal vision. In 1 case complete clearing of the cornea occurred after eighteen months of vitamin therapy although only perception of light was present in one eye and perception of fingers in the other when therapy was begun. Absorption of corneal exudates and return of normal vision took place in another case after four weeks of vitamin therapy alone.

Riboflavin when administered alone or in combination with vitamin E was effective primarily in relieving some of the photophobia and reducing the extent of circumcorneal injection and capillary proliferation. It appeared to have no effect, however, on the rate of absorption of corneal opacities and scars.

It is suggested that vitamin E combined with vitamin B complex is a most valuable adjunct in the treatment of interstitial keratitis. It hastens the absorption of corneal exudates and opacities of long standing, it prevents further organization of scar tissues of the cornea and it reduces excessive capillary permeability. The addition of riboflavin to the vitamin E is believed to enhance the action of the former by its beneficial effect on cellular oxidative processes in the cornea. If the patient has received ample treatment in the past, antisyphilitic therapy is apparently not needed to produce complete disappearance of the visual symptoms and corneal opacities of interstitial keratitis. Artificial fever therapy is of value mainly in preventing relapses and in producing more rapid amelioration of acute symptoms. It has little effect when administered alone on the rate of absorption of corneal opacities of long standing.

NOTE.—Since this paper has been submitted for publication a second male patient with severe bilateral interstitial keratitis in a state of relapse has been placed under vitamin E and B therapy while chemotherapy is being temporarily withheld. This patient failed to show any improvement under sulfathiazole therapy and following intravenous injections of riboflavin, 5 mg. daily. The clearing of the corneas has been slower in this case than in the female patients similarly treated, although the rate of improvement has been slightly more rapid than during the previous attack, when he received chemotherapy and local therapy.

Dr. Walter B. Rahmanop, ophthalmologist at the Elliot Hospital, aided in the visual check-up and follow-up examinations of 4 patients. Eli Lilly and Company, through Dr. C. G. Weigand, supplied some of the wheat germ oil and mixed tocopherol capsules used in this investigation. Mixed tocopherol capsules were also supplied by the Winthrop Chemical Company, Inc., and the vitamin B complex was made available by Dr. J. M. Ruegsegger, of the Lederle Laboratories, Inc.

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PATHOLOGIC ANATOMY OF MYOPIC EYE WITH REGARD TO NEWER THEORIES OF ETIOLOGY AND PATHOGENESIS OF MYOPIA

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Although the pioneer work of Steiger [1] has considerably enlarged the present conception of the development of the different types of refractions of the eye, there remain a number of problems in this field awaiting solution. It seems that starting from infantile hyperopia the development of the eye follows a plan of which the aim is to reach a state of approximate emmetropia. A definite correlation of growth between the various parts of the bulbus is necessary in order to accomplish this aim. Eyes with high refracting power develop a short axis, and eyes with low refraction develop a longer axis (Wibaut,[2] Straub,[3] Wessely [4]). Thus emmetropia does not depend either on an axis of a certain length or on a certain power of the refracting system but is the result of an adequate correlation of the two. This becomes obvious when one takes into consideration the fact that the average length of the emmetropic eye varies from 22 to 25 mm. (Wessely). Besides emmetropia there exists ametropia on both sides, with hyperopia on one and myopia on the other, which must not be considered pathologic but a result of normal variability. It has been demonstrated that if one draws a curve to show the number of instances of emmetropia or any degree of ametropia found among a certain number of cases, the peak is at 0.50 D. of hyperopia. This means that the greatest number of persons are nearly emmetropic. Nevertheless, there is a definite difference between the shape of the curve based on statistics and the binominal type of curve which would be expected if all ametropia were merely the result of biologic variability.

In the sector of the curve covering higher grades of myopia the broadening is particularly impressive. It can be shown that the curve becomes fairly regular when one does not take into consideration cases in which there is more than 6 D. of myopia (Tron [5]) or all of those in which there are alterations in the fundus, for instance conus (Betsch [6]). But even if one does not take into consideration extreme variations the curve appears too long, or, in other words, there are many more cases around the optimum (+ 0.50 D.) than would be expected from purely mathematical calculations. It is not possible to answer by scientific methods the

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6. Betsch, A.: Ueber die menschliche Refractionskurve, Klin. Monatsbl. f. Augenh. **82**: 365, 1929.

question of why there are so many more cases of normal refraction than would be expected from calculations. One is facing here one of the mysteries of creation.

It is a fairly well established assumption that in each person the refraction of the eye is determined to a large extent by hereditary factors (Waardenburg [7]).

Having taken into consideration these facts, one might establish the limit of physiologic variations at about 6 D. of myopia. Higher grades, especially when accompanied with destructive alterations in the fundus, ought to be considered pathologic with correlation having been lacking in the development of the various parts of the bulbus.

The present study involves a consideration of these grades of high myopia.

It is known that the length of the axis is the most important factor in determining the grade of such myopia. But it has been found by Schnabel and Herrnheiser [8] that in myopia of 2 to 8 D. the length of the axis varies between 23 and 26 mm., the range for emmetropia being 22 to 25 mm. In the high grades, however, the length varies from 27 to 36 mm. (Wessely.[4]). For this reason such grades have always been called axis myopia. This spectacular fact of lengthening of the axis led to the assumption of a stretching process which produced both the length of the eye and the alterations observed ophthalmoscopically and histologically in the fundus. Some anatomic findings, for instance the thinning of the sclera at the posterior pole, supported this theory, and once the idea was started all other alterations found in the myopic fundus were attributed to this stretching factor. Although this theory has become an almost universally accepted basis for all explanations of myopia, one must acknowledge that the existence of such a stretching factor has never been demonstrated. However, it has been claimed that the stretching is dependent on the intraocular tension.[9]

The intraocular tension is by no means elevated in the myopia in comparison with the emmetropic eye but has, on the contrary, been found reduced in about 25 per cent of cases of myopia of the higher grades (Wolff [10]). To meet this objection the theory was proposed that there might be a weakness of the sclera toward normal or even subnormal intraocular pressure. A statement made by Lange [11] pointing in the same direction, according to which the sclera of myopic eyes should contain fewer elastic fibers than the normal sclera, has been entirely refuted by Birch-Hirschfeld's [12] findings. Kuschel [13] went so far as to claim a relation between flatfoot and myopia as a sign of weakness of all supporting tissues. However, such weakening of the sclera would probably lead to a general enlargement, or buphthalmos. Since buphthalmos develops only during the period of general growth, this theory leaves unexplained the late development of or increase in myopia. It is known, moreover, that growth of the bulbus is generally complete at about the age of 15, an age at which the myopic process not only is active but may be accentuated.

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12. Birch-Hirschfeld, A.: Zur Frage der elastischen Fasern in der Sclera hochgradig kurzsichtiger Augen, Arch. f. Ophth. **60**:552, 1905.

13. Kuschel, J.: Die Erschlaffung der Koerperkonstitution als Veranlagung zur Kurzsichtigkeit, Ztschr. f. Augenh. **51**:339, 1923.

So the theory was evolved that the weakness of the sclera is confined to the posterior pole and that the other layers, the choroid and retina, follow the enlargement there and become stretched. But Vogt [14] previously pointed out that the alterations in the choroid and in the retina do not occur when this so-called stretching is supposed to happen, that is, when the myopic bulbus becomes oblong, but sometimes many years later. He therefore opposed this purely mechanical hypothesis and tried to find an explanation with a biologic basis. He took as his major premise the fact known in embryology that the principal part of an organ always determines its size and shape and that the surrounding protective tissues accommodate themselves to that shape, just as the size of the brain determines the size of the skull. So the retina, which undoubtedly may be considered the predominant functioning element of the optic apparatus and is indeed but an outpost of the brain, may determine the size of the bulbus. A retina with much inherent growing power, therefore, would predetermine a long eyeball and produce myopia, while a retina with less growing power might cause the bulbus to be small, with emmetropia or hyperopia. There are facts which seem to prove a relation between the size of the eye and the development of the brain. I [15] have found that hyperopia is more likely to be found in children with poor intelligence. On the other hand, Braun [16] saw higher efficiency in myopic than in average children. Furthermore, the eye increases from one to three and four-tenths times from birth to maturity (the brain from one to three and seven-tenths) whereas the figures for the entire body are only one to one and seven-tenths according to Wessely.[4] I am studying the problem along this line also, but the aim of the present study is to deal exclusively with the pathologic anatomy of myopia.

I shall try to explain the pathologic anatomy in the myopic eye without relying on the stretching theory. I also shall try to ascertain whether the growing power of the retina is the determining factor in the pathogenesis of myopia or whether other possibilities must be taken into consideration.

It may be useful to begin with a recapitulation of the most important anatomic changes which are generally considered to be characteristic of myopia. They are:

1. Lengthening of the axis of the bulbus, enlarging of the posterior part of the bulbus and thinning of the sclera in the posterior segment accompanied by a lamellar arrangement of the scleral fibers.

2. Anomalies at the optic disk such as an oblique entrance of the optic nerve into the globe, myopic crescent, supertraction of the nerve fibers on the nasal side, temporal loop of the nerve fibers and lacunal atrophy of the optic nerve.

3. Atrophy of the choroid and changes in the ciliary muscles.

4. Changes in the retina, such as atrophy of the pigment epithelium, rods and cones; cystic degeneration, and total atrophy.

The chronologic appearance of these changes is interesting and is of special importance in relation to the retina, as shall be seen in the discussion of the various theories. In the discussion of Vogt's [14] paper, there emerged the suggestion that it would be most valuable to examine eyes in the early stages of myopia in order to find out where the first changes can be seen, as later on, when the choroid

14. Vogt, A.: Zur Genese der sphaerischen Refractionen, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch., 1922, p. 68.

15. Stocker, F.: Ueber Beziehungen zwischen Refraction und Gehirnentwicklung (Ein Beitrag zum Refractionsproblem), Arch. f. Ophth. **133**:131, 1934.

16. Braun, E.: Beitrag zur Frage von den Beziehungen der Refraktion der Augen zur Gehirnausbildung, Schweiz. med. Wchnschr. **65**:1124, 1935.

has become atrophic, the retina of course suffers, the external layers of the retina being nourished by the choroid. It is obvious that eyes in the early stages of myopia are difficult to get for anatomic examination.

While examining microscopically a great number of myopic eyes from the collection of the Institute of Ophthalmology of the Presbyterian Hospital and some from the New York Eye and Ear Infirmary, I had a chance to study histologically a myopic eye from a 15 year old boy whose second eye showed myopia of 2.75 D. with typical myopic changes in the fundus. The eye had been enucleated because of severe blunt trauma with intraocular hemorrhage but without rupture of the bulbus.

The bulbus was much elongated, especially in the posterior segment, and differed decidedly in shape from a bulbus with buphthalmos due to rise of tension during growth. The accompanying table shows the measurements of this eye in comparison with a normal eye of the same age from the collection and with the average emmetropic and myopic eye according to Wessely.[4]

The following statement seems justified after a comparison of the figures in this table:

The axis of the eye of the young myopic patient was markedly longer than that of the average emmetropic eye and of the eye of a normal 15 year old boy. Furthermore, its sclera at the posterior pole appeared to be thinner than in the average emmetropic eye. Near the ora serrata the sclera was about as thick as it is in the average emmetropic eye and somewhat thicker than in the average myopic eye and the eye of a normal 15 year old boy. These

Comparative Measurements of a Myopic Eye from a 15 Year Old Boy

	Length of Axis, Mm.	Thickness of Sclera, Mm.		
		Posterior Pole	Equator	Oro Serrata
Emmetropic eyes (average according to Wessely)..	22-24	0.66	0.42	0.53
Myopic eyes (average according to Wessely).....	27-35	0.21	0.32	0.48
Normal eye from 15 year old boy.....	22	0.41	0.30	0.34
Myopic eye from 15 year old boy.....	28	0.34	0.41	0.46

findings lead to the conclusion that an enlargement of the bulbus had taken place, with the abnormal growth in length confined to the posterior segment. This is to be considered typical of myopia, in contradistinction to buphthalmos, in which the enlargement is uniform throughout the bulbus.

Microscopic analysis revealed, besides signs of intraocular hemorrhage and a slight cupping of the optic nerve, the following alterations typcial of myopia:

1. Sclera: The structure of the sclera was little different from that in a normal specimen and did not show in any degree the flat arrangement of the fibers reported by several authors to be typical of high myopia. I myself have confirmed this arrangement in an eye with high myopia with an axis of 30 mm.

2. Optic disk: Neither the choroid nor the retina reached the temporal border of the optic disk. The sclera, temporally from the disk for a short distance, lay directly beneath the nerve fiber layer of the retina. Then, sharply defined, the almost normal choroid and retina appeared. There was no partial atrophy of the choroid, which sometimes extends farther temporally in cases of pronounced myopic conus. Nevertheless, there was no question of the presence of a beginning crescent. Because of an artificial defect in the specimen the real position of Bruch's membrane in that area was not well defined. On the nasal side the retina and choroid clearly overlapped the border of the optic disk, the choroid going about as far as the retina. The nerve fibers turning around the edge of the retina reached the nasal side of the optic nerve and formed a loop. The picture was typical of so-called supertraction, a phenomenon which according to my reasoning would better be called supervolution, as will be explained later. The so-called temporal loop of the nerve fibers in the area of the crescent was absent (fig. 1).

3. Choroid: The choroid was normal throughout. The blood vessels were of normal size and number in all three layers. Extensive regularly distributed pigmentation was present. Bruch's membrane was nowhere interrupted. The suprachoroid area did not show any change. Not the slightest sign of atrophy could be detected.

4. Retina: The retina was normal throughout. The pigment epithelium and the rods and cones showed no changes. No cystic degeneration (Blessig's cysts) was present. There were no signs of thinning, nor did counting of the nuclei in the different layers according to Ida Mann's [17] method reveal any change. A section through the macula as seen in figure 2 showed the absolutely normal condition of choroid and retina in that area.

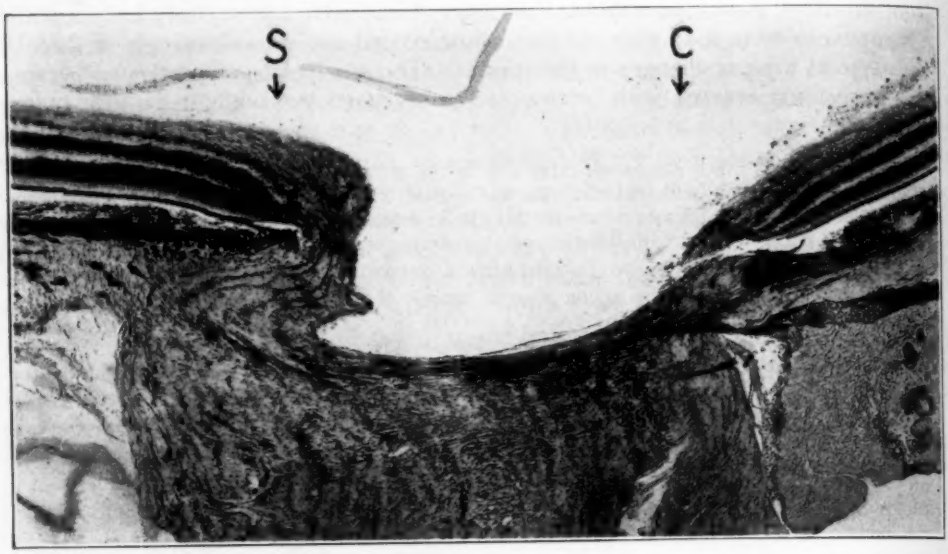

Fig. 1.—Optic nerve head of a 15 year old myopic patient. *C*, crescent; *S*, supratraction, or supervolution.

Fig. 2.—Region of the macula of a 15 year old myopic boy. Note the perfectly normal structure of the retina and choroid.

17. Mann, I.: The Development of the Human Eye, New York, The Macmillan Company, 1928.

To summarize, the following anatomic changes typical of myopia were present: abnormal length of the bulbus, slight thinning of the sclera at the posterior pole, beginning temporal crescent and supertraction of the retina on the nasal side of the disk. Absent were all signs of atrophy in the choroid and the retina. It therefore may be stated that this juvenile myopic eye was already considerably larger than a normal emmetropic eye. The lengthening was accompanied by a slight thinning of the sclera at the posterior pole combined with beginning temporal crescent, no other signs of stretching being present.

I shall now leave discussion of this eye in order to review the pathologic changes in myopia in general as reported in the literature and as illustrated by my study of numerous myopic eyes. Special attention is given the question whether these changes are observed exclusively in myopia.

1. Thinning of the sclera at the posterior pole: This can be found in higher grades of myopia almost regularly and has been observed in many specimens. As the case reported reveals, it may be present at the beginning of the myopic lengthening process and therefore be considered an early sign of myopia. On the other hand, E. Fuchs [18] and others have seen myopic eyes with a sclera even thicker than that in the average emmetropic eye. Extreme myopia, however, was not present among these eyes. A flat arrangement of the scleral fibers is generally found in cases of thinning. No parallel to these scleral changes has been found in senility.

2. Temporal conus, or crescent: A temporal crescent is seen frequently in high myopia both ophthalmoscopically and microscopically. But the fact that there is no inevitable relationship between the degree of myopia and the size of the crescent has been emphasized by Parsons [19] and others. Although more frequent in myopic eyes, crescents are found in emmetropic and even in hyperopic eyes. The pathogenesis of the crescent, or conus, has been explained by an eversion of the sclerotic canal of the optic nerve (Stilling [20]) and by atrophy of the choroid following stretching. Evidence for the first possibility can be seen in those cases in which repeated ophthalmoscopic examinations over a period of several years reveals the migration of a pigment spot temporally from the border of the disk as the formation of the conus progresses. Many variations can be observed in the relation between the retina and the choroid at the site of the crescent. Sometimes they remain the same distance from the disk, and sometimes the atrophy of the choroid goes much farther, being overlapped by normal retina or by retinal layers which persist in the area of the crescent. Bruch's membrane may persist well toward the disk or, conversely, may terminate well back of the retinal border (fig. 4). It is hard to say whether or not in the latter case the cause is choroidal atrophy. In this connection one should bear in mind the fact that a crescent similar to that found in myopic eyes can be seen in old eyes which are not at all myopic, as shown by Buecklers,[21] and that the crescent is not always located on the temporal side but can be circumpapillar on the nasal or the inferior side of the optic disk. Von Szily [22] found that 13.43 per cent of all crescents are not temporal ones, and he stated the belief that a tendency toward conus develops in fetal life. The pictures he reproduced from fetuses indeed resembled the ordinary crescent. It should be said, however, that conus has rarely been observed in early life, generally not appearing until the second decade. And there is no evidence that the crescent-

18. Fuchs, E.: Myopische Augen mit dicker Sclera, Klin. Monatsbl. f. Augenh. **62**:429, 1919.
19. Parsons, J. H.: The Pathology of the Eye, New York, G. P. Putnam's Son, 1909.
20. Stilling, J.: Ueber den Conus, Ztschr. f. Augenh. **4**:563, 1900.
21. Buecklers, M.: Anatomische Untersuchungen ueber die Beziehungen zwischen der senilen und der myopischen circumpapillaeren Aderhautatrophie, Arch. f. Ophth. **121**:243, 1929.
22. von Szily, A.: Ueber den "Conus in heterotypischer Richtung," Arch. f. Ophth. **110**: 183, 1922.

like formations found in embryos would have been crescents in adults. I have a feeling that they might as well be considered transitory stages of development. Combined with the conus in many cases is the following peculiar phenomenon:

3. Temporal nerve fibers loop: In cases of crescent in which the retina overlaps the choroid toward the optic disk, the nerve fibers corresponding to the most

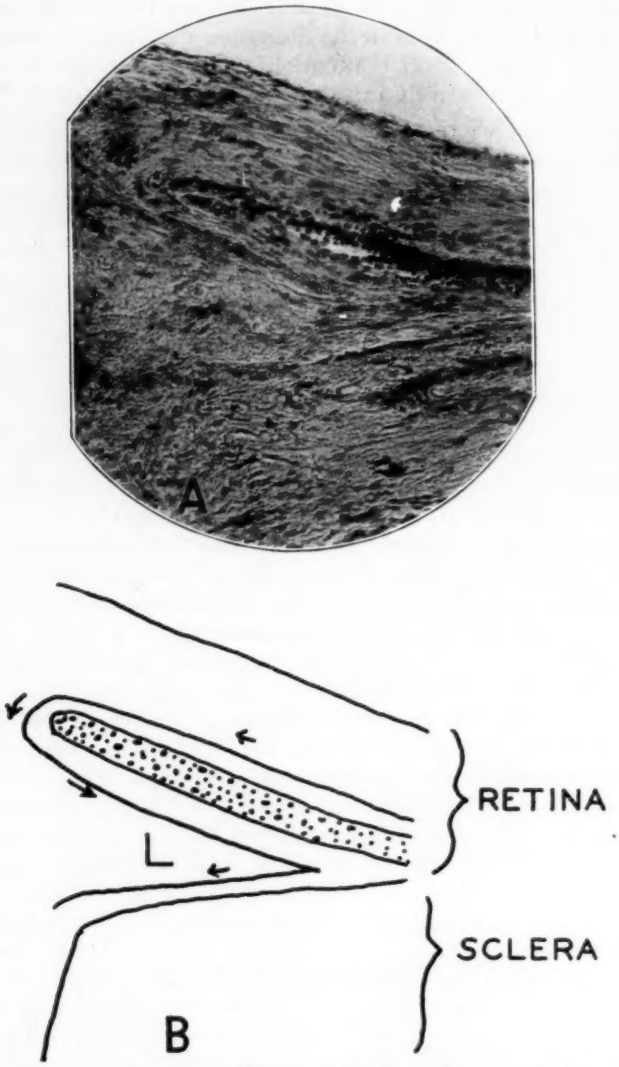

Fig. 3.—*A*, temporal part of the optic nerve head of a 66 year old myopic patient, with crescent and nerve fiber loop. The choroid is absent in the area reproduced. *B*, schematic explanation of *A*; *L* indicates the nerve fiber loop.

temporal part of the optic nerve happen to turn around the edge of the end of the retina, following a direct retrograde direction beneath the retina to a certain point, where they turn back in a sharp angle in order to reach the optic nerve. Figure 3, showing a section of a 66 year old myopic eye, reproduces this phenomenon photographically as far as I know for the first time, the previously published pictures

having been drawings. This anatomic peculiarity was described first by Weiss [23] and was studied extensively by Heine.[24] The latter stated the belief that while drawn back by the stretching process Bruch's membrane by means of its ramifications extending into the optic disk pulls along some of the nerve fibers. But Siegrist [25] and Inoye [26] found that there is no evidence of such ramifications. In my investigation of Bruch's membrane I found that its site in the normal eye relative to the choroid and optic disk is subject to great variation. Sometimes it reaches far into the optic disk, going much farther than the choroid. Sometimes it stops at the border of the disk, together with the choroid. The fact that the nerve fiber loop sometimes does not reach the end of the membrane (fig. 4) is one more indication of the improbability of Heine's explanation. Siegrist [25] gives another explanation. He could demonstrate fibers from the connective tissues of the choroid spreading

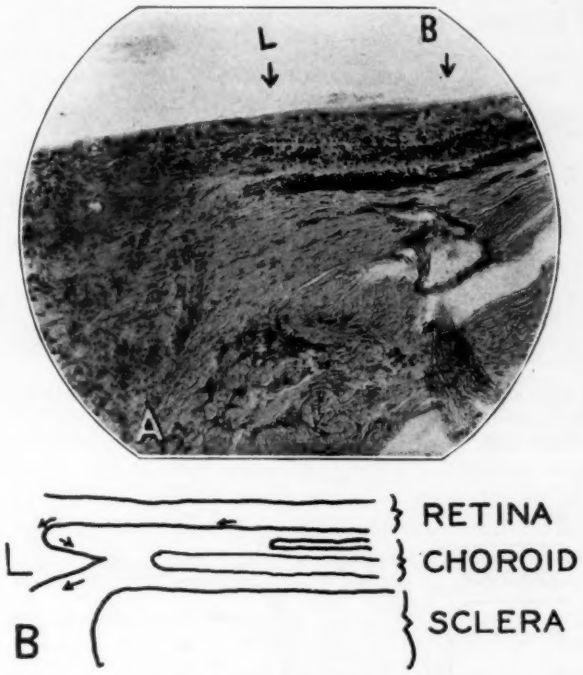

Fig. 4.—*A*, temporal part of the optic nerve head of a 66 year old myopic patient; *L* indicates the nerve fiber loop and *B* the termination of Bruch's membrane. *B*, schematic explanation of *A*; *L* indicates the nerve fiber loop.

out into the optic nerve head. He stated the belief that when the choroid is retracting after the stretching process these fibers may pull with it some of the nerve fibers in the neighborhood. As will be seen later, the course of the neuroglia fibers of the retina shown with Masson's trichrome stain (hematoxylin, fuchsin and aniline blue) leads me to an explanation which appears more natural. I

23. Weiss, L.: Zur Anatomie der Eintrittsstelle des Sehnerven, Period. internat. Ophth.-Cong. Ber. **7**:339, 1888.

24. Heine, L.: Beiträge zur Anatomie des myopischen Auges, Arch. f. Augenh. **38**:277, 1899.

25. Siegrist, A.: Refraktion und Akkommodation des menschlichen Auges, Berlin, Julius Springer, 1925.

26. Inoye, S.: Ueber die sog. Auffaserung der Membrana elastica chorioideae im Sehnervenkopf und ihre Rolle bei der myopen Konusbildung, Klin. Monatsbl. f. Augenh. **74**:124, 1925.

wish to emphasize, however, that I observed the nerve fiber loop also in an eye with senile crescent which did not show any signs of myopia. Previously Wessely [6] pointed out that it may occur in nonmyopic eyes.

4. Supertraction of the retina over the nasal part of the disk: Often in high myopia the retina overlaps the nasal part of the disk. As demonstrated by the 15 year old eye described, it can occur in an early stage. In this eye the choroid also took part in the overlapping. This of course is not the rule. Often the choroid stops at the border of the disk and the retina alone extends farther, as shown in figure 5. Therefore the explanation given by A. Fuchs [27] seems not to be accurate. He said:

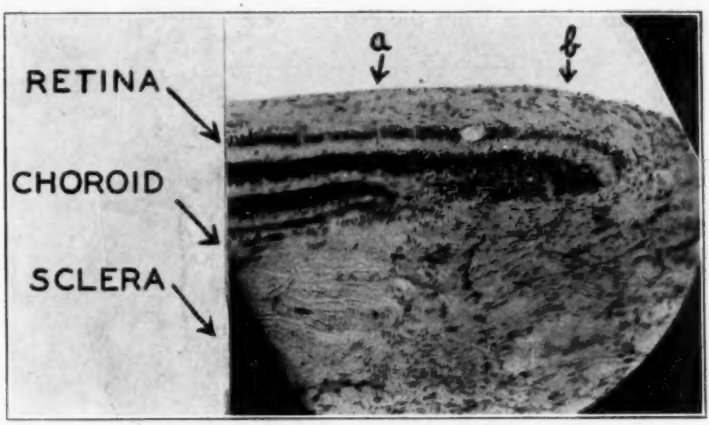

Fig. 5.—Nasal part of the optic nerve head of a myopic eye, showing supertraction, or supervolution. The choroid stops at the margin of the optic disk (*a*), while the retina partly overlaps the nerve head (*b*).

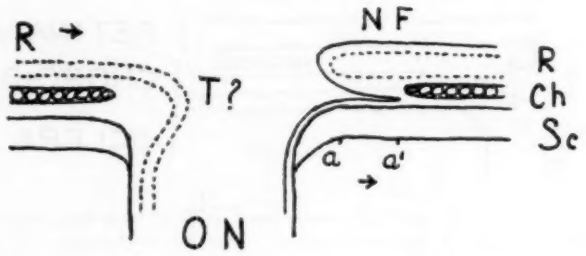

Fig. 6.—Myopic crescent, nerve fiber loop and supertraction shown schematically. *R*, retina; *Ch*, choroid; *Sc*, sclera; *ON*, optic nerve; *NF*, nerve fiber loop; *T*, point where hypothetical power would have to exercise traction to produce supertraction; *aa'*, area of crescent where the sclera is not covered by the choroid.

In cases of progressive myopia the membranes of the eye are distended more and more at the posterior pole of the eye, the distention of the sclera being greater than that of the choroid. Consequently the opening in the choroid through which the optic nerve passes is displaced temporally to the opening in the sclera so as to pull the choroid over the nasal margin of the scleral canal.

To explain this mechanical problem I drew a little sketch (fig. 6). It shows schematically the relative position of the optic nerve, choroid and retina in case of supertraction in which the choroid is not involved in the process. It is obvious

27. Fuchs, A.: Atlas of the Histopathology of the Eye, Leipzig, F. Deuticke, 1924.

that the force of the traction should apply to point T, but it is hard to imagine what pulling power would act and how it could act there. I therefore prefer the purely descriptive expression "superposition," which implies nothing relative to the supposed mechanism (Wessely [4]). I shall, however, describe later another tentative theory which would rather lead to the expression "supervolution." Although the phenomenon of the superposition is apparently found rarely in conditions other than myopia, it is not absolutely confined to myopia. Verhoeff [28] observed it in a case of amaurotic idiocy in which myopia was certainly absent. But this combination, on the other hand, seems not to be characteristic either. In 2 cases of amaurotic idiocy represented in the collection of the Institute of Ophthalmology of the Presbyterian Hospital I could not find it.

5. Atrophy of the choroid: It cannot be the purpose of this study to enumerate all details of the choroidal atrophy associated with myopia. One must keep in mind the fact that in the juvenile myopic eye the choroid may be without any signs of atrophy although the eye has grown considerably in length. This statement is supported by the anatomic picture in the case reported. Vogt [29] was certainly right when he pointed out that the atrophy of the choroid does not occur at the time of the lengthening of the bulbus but often many years later. It also must be noted that there can be a great resemblance between some choroidal changes found in myopic eyes and senile degeneration. No anatomic observations have so far been published which would support Lindner's [30] theory that a defective suprachoroid space would deprive the sclera of protection against the hypothetic digestive action of the tissue. I carefully examined the slides of the macular region in the case reported but could not find any signs of such deficiency.

6. Retinal changes: One of the alterations often found in myopic eyes is cystic degeneration at the ora serrata. This is not, however, regularly found in myopic eyes, nor is its appearance confined to myopia. In the juvenile eye described no Blessig's cysts could be seen. These cysts seem to be present more frequently in old age, but, on the other hand, they cannot be considered typically senile. In the material at hand I found them in normal eyes of patients 15, 27 and 47 years of age. Ochi [31] has observed cystic degeneration in children under 4 years. Kalmer [32] stated that Blessig's cysts are so frequently unrelated to a specific condition that they can hardly be considered pathologic.

I could not find other types of degeneration, such as disappearance of the pigment epithelium, crippling of rods and cones and, finally, atrophy of all the layers, except in cases of advanced involvement in which the respective parts of the choroid were definitely deteriorated. I never found retinal atrophy over a sound part of the choroid.

7. Lacunar atrophy of the optic nerve: This severe condition of the optic nerve stem is found in high myopia combined with extensive destruction of all the tissues of the bulbus. But it is also found in advanced stages of glaucoma, and there is some controversy about whether it can be found in simple myopia

28. Verhoeff, F. H.: Amaurotic Family Idiocy: Histological Examination of a Case in Which the Eyes Were Removed Immediately After Death, Arch. Ophth. **38**:107, 1909.

29. Vogt, A.: Ueber Beruehrungspunkte der senilen und myopischen Bulbusdegeneration, Klin. Monatsbl. f. Augenh. **32**:212, 1924; footnote 14.

30. Lindner, K.: Neue Gedanken ueber die Entstehung der Kurzsichtigkeit, Klin. Monatsbl. f. Augenh. **103**:582, 1939.

31. Ochi, S.: So-Called Cystic Degeneration in the Peripheral Retina, Am. J. Ophth. **10**:161, 1927.

32. Kalmer, W.: Haut und Sinnesorgane, in von Möllendorf, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1936.

alone or combined with glaucoma. At any rate, it cannot be considered typical of myopia.

In view of the facts regarding the pathologic anatomy of myopia derived from a careful study of the literature and from my observations, the issue of this study may now be considered.

There are two main questions. The first one concerns the pathogenesis of the supposed stretching process of the myopic eye. The second is, Is it necessary to admit something like a stretching process in order to understand the pathologic anatomic changes or can these be explained as well or better by another theory?

The important fact derived from study of a juvenile myopic eye is that the sclera can be thinned and the bulbus considered enlarged at the posterior pole while neither the choroid nor the retina shows the slightest pathologic change. This makes it hard to believe that the alterations found in the choroid and retina in older myopic eyes are the result of stretching. One would expect the stretching effect on the choroid and retina to appear when the enlargement of the bulbus occurs, whereas the choroidal and retinal atrophy begins much later, when the size of the bulbus has generally become more stable.

These facts are much easier to understand if one follows Vogt's [14] theory that the retina is the determinative factor in the size of the eye. If the retina has a stronger tendency to grow than the sclera, the latter might be stretched. This

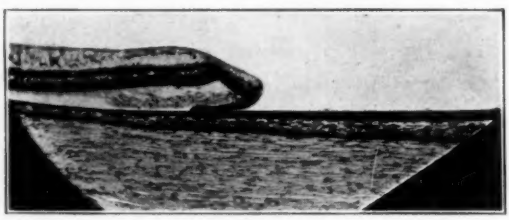

Fig. 7.—Fold of retina near the ora serrata in the eye of a newborn infant.

"push" of the retina on the sclera has, of course, to be taken not as mechanical but as purely biologic. Besides the fact that in a young myopic person the sclera is already affected while the retina is intact, other features of the anatomy of the myopic eye can be explained naturally by the theory. One of these is the supertraction of the retina over the nasal part of the optic disk. It should be noted that the way of postembryonic growth of the retina is not yet well known. It is the opinion of many authors that the retina does not produce new cells after birth. If there is any postembryonal growth it is to be expected at the ora serrata according to Mann [17] and Kalmer.[32] Whether the retinal folds I found near the ora serrata in many eyes of newborn infants have something to do with this question I do not know (fig. 7). But, although Lange,[33] who first described these folds as regularly present in the newborn, later was inclined to take them for an artefact due to fixation, the fact remains that they can never be seen in adults although the same methods of fixation are employed. One could imagine that the young tissue, still growing, in the region of the ora might be especially sensitive to the fixation chemicals and therefore give origin to these folds. If in myopia the growing retina is the determinative factor the new retinal tissue produced at the ora would push the existing retina backward. Obviously the sclera gives way

33. Lange, O.: Ber. ü. d. Versamml. d. ophth. Gesellsch., 1893, p. 236; Anatomie des Auges des Neugeborenen: Suprachoroïdalraum; und sog. physiologische Excavation der Sehnervenpapille, Klin. Monatsbl. f. Augenh. **39**:202, 1901.

to this biologic pressure at the posterior pole, becoming thinned, and the bulbus enlarges, whereas the retina, as the acting factor, does not show signs of stretching but follows the receding sclera. It is known that the sclera does not give way as much on the nasal side of the optic disk as it does on the temporal side. The growing retina pushes ahead over the nasal part of the disk, thus leading to the condition known as supertraction or superposition. As to the described mechanism's being more a push than a traction, I would call it rather supervolution (figs. 6 and 8) depending on the inherent growing power of the retina and the choroid. The latter may take part in the overlapping or let the retina slide over. Thus it can be explained why sometimes the retina alone (fig. 5) and sometimes both the retina and the choroid are present (fig. 1) in the supertraction.

On the temporal side of the disk the opposite happens. There the retina finds ample space to grow because of the receding of the sclera. It may in certain cases happen that the sclera responds to the biologic pressure of the retina in an exaggerated way, so that the growing ability of the retina and choroid becomes insufficient to compensate for the enlargement of the sclera, the result being a

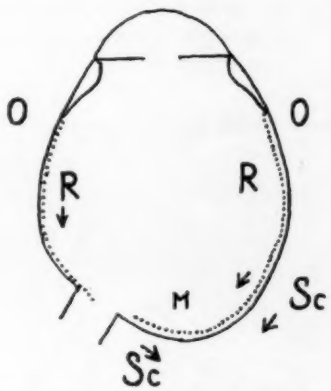

Fig. 8.—A drawing to show how the retina (*R*) would grow backward from the ora serrata (*O*), causing a distention of the sclera (*Sc*) at the posterior pole (*M*, macula) and overlapping of the nasal part of the optic disk: supervolution.

gap between the end of the choroid and retina and the temporal border of the disk. This will lead to the condition known as temporal crescent, or conus. The crescent therefore will be due to eversion of the scleral canal of the optic nerve on the temporal side and incongruence of growth of the choroid and retina with growth of the sclera. This may become complicated later by atrophic degeneration. If this assumption is correct the peculiar phenomenon of the temporal nerve fiber loop can be explained naturally in the following way: In figure 9, which shows a section through the optic disk of a normal bulbus, stained with Masson's trichorome stain, it can be seen clearly that at the edge of the disk a bundle of neuroglia fibers turns from the retina temporally into the inner layers of the sclera. (These fibers in reality appear red.) As the nerve fibers necessarily are interlocked with the neuroglia fibers, some of them must be drawn along with the neuroglia if eversion of the temporal side of the sclerotic canal occurs. This explanation seems to be more natural to me than a pull by choroid fibers radiating into the nerve head (Siegrist [25]) or even by Bruch's membrane (Heine [24]).

After a consideration and evaluation of all the facts discussed the following explanation of the pathogenesis of the myopic enlargement of the bulbus and the accompanying anatomic changes is possible:

Under the assumption that the enlargement of the bulbus is due to a stretching process it appears most likely that the determinative factor is the retina. During the initial stages of the development of myopia the retina remains normal, whereas the sclera becomes thinner at the posterior pole as the bulbus enlarges. Later atrophic changes in the choroid take place, and only after that the retinal atrophy occurs. Briefly, the pushing part naturally is not stretched, whereas the pushed sclera, having to adapt itself to the larger size of the pushing part, becomes thinner, and the arrangement of its fibers shows a change which can possibly be explained by a stretching process. The fact that the retinal vessels are often seen to be stretched out in high grade myopia does not prove a stretching of the retina proper, as embryologically the blood vessels are not of the same neuroectodermic origin. At the time when the retina begins to show atrophy there is generally no longer any considerable stretching under way. The retinal changes appear exclusively in areas where the choroid is deficient. As the choroid is supposed to supply the outer layers of the retina, its deterioration may cause damage to the latter. Thus the retinal atrophy should be considered as a secondary happening.

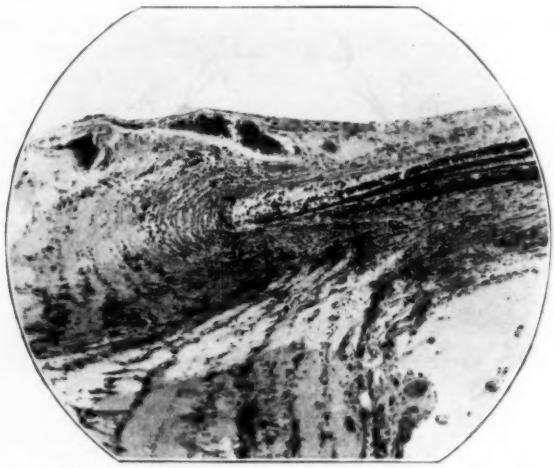

Fig. 9.—Temporal part of the optic nerve head of a normal eye prepared with Masson's trichrome stain. Neuroglia fibers of the nerve fiber layer of the retina turn around the end of the retina and reach the superficial layers of the sclera.

But the assumption of a stretching process is not essential to explain the pathologic anatomic changes in myopia, and the fact that the same or very similar alterations can be found in various conditions other than myopia makes it doubtful. It seems to be possible that each coat of the bulbus, retina, choroid and sclera has its own inherited potential of growth. In the majority of persons there exists enough coordination to secure the development of an eye which is emmetropic or nearly so. In myopia there would be incongruence of growth of the different constituents, not only resulting in an abnormally long bulbus but producing the various anatomic phenomena observed in myopia. An inherited degenerative factor may produce chorioretinal atrophy similar to senile changes. Most probably myopia and its anatomic attendants are not a hereditary unity but a combination of individually varying factors which may or may not result in a manifestation of myopia. This, of course, may explain the difficulty of establishing the definite course of heredity in myopia.

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COMPARISON OF A NEW SENSITOMETRIC METHOD WITH USUAL TECHNICS OF REFRACTION

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AND
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During the evolution of a new technic of subjective refraction various aspects and results have been presented.[1] In the present paper comparisons are made of this sensitometric method with the usual technics of refraction from various static and dynamic viewpoints. The apparatus in its present form is illustrated in figure 1. The knurled knob at the right operates the circular gradients of a specially adapted Luckiesh-Moss visibility meter, and the operator conveniently reads the settings, made by the subject, on the enclosed cylindric scale at the left. The position of the convergence target is shown for a test of near vision. Various details of the apparatus and technic are omitted here, as they have been presented heretofore.[1a]

SYNOPSIS OF METHOD

The sensitometric is a subjective method of refraction in which the dioptric power producing maximal visibility is determined with the Luckiesh-Moss sensitometer.[1a] The measurements of visibility are derived from brightness contrast rather than from acuity thresholds. By means of a convergence target of sufficient size and indistinctness, as illustrated in figure 2, the visual thresholds are determined with binocular convergence in force but in the proved absence of any adequate optical stimulus for relative accommodation. This new technic also differs from the usual subjective tests in that identical procedures are involved in both static and dynamic tests. Thus the data obtained under conditions of near and of distant vision are directly comparable. In a detailed laboratory study of 20 cases and a clinical study of 100 cases, the method indicated (1) that for the typical emmetropic subject, the eyes in the so-called "position of rest" are accommodated for a point anterior to the retina to the extent of about 0.75 D. and (2) that for such a subject there is no evidence of a lag of accommodation within the range of habitual near vision.

STATIC REFRACTION

Control of Relative Accommodation.—This compensatory function is avoided in the sensitometric procedures by elimination of all adequate optical stimuli for accommodation from the entire binocular field.[1b] The attainment of this essential control is experimentally proved by symmetric relationship between visibility and

†Lieutenant Commander Moss died Feb. 14, 1943.

From Lighting Research Laboratory, General Electric Company.

1. Luckiesh, M., and Moss, F. K.: (*a*) New Method of Subjective Refraction Involving Identical Technics in Static and Dynamic Tests, Arch. Ophth. **23**:941 (May) 1940; (*b*) The Avoidance of Dynamic Accommodation Through the Use of a Brightness-Contrast Threshold, Am. J. Ophth. **20**:469 (May) 1937; (*c*) The Plus-Power Bias in Static Refraction, Am. J. Optom. **18**:313 (July) 1941; (*d*) Functions of Relative Accommodation, Am. J. Ophth. **24**:423 (April) 1941; (*e*) A New Method of Subjective Refraction at the Near-Point, Am. J. Optom. **18**:249 (June) 1941; (*f*) Initial and Residual Effects of Ophthalmic Prisms on Visibility and Accommodation, Arch. Ophth. **29**:968 (June) 1943.

refraction about the dioptric axis of maximal visibility. In other words, over and under additions of equal dioptric powers blur the vision to equal degrees.

In the fogging method of refraction, it is probable that relative accommodation is avoided in the initial phases of the examination. However, the stimulus for relative accommodation increases in intensity as the plus power is reduced, and this function may be influential in the final stages of the fogging test. Furthermore, it is not certain that both positive and negative relative accommodations are completely avoided in these procedures, particularly when an exact correction is sought.

Control of Convergence.—The sensitometric method involves binocular parafoveal stimuli of sufficient intensity to insure convergence at a point in the plane

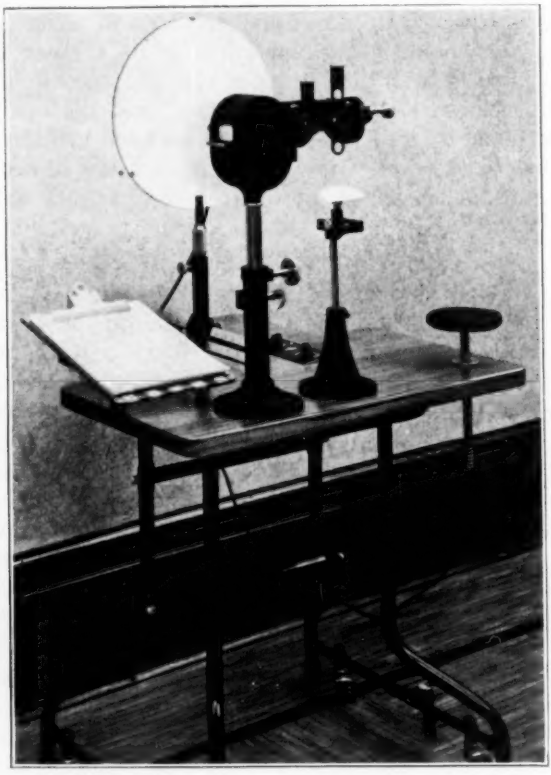

Fig. 1.—The Luckiesh-Moss sensitometer in its latest form. The position of the convergence target is shown for a test of near vision.

of the test object.[1a] The blurred luminous stimuli for convergence have been shown to be inadequate stimuli for relative accommodation. In the usual methods of examination, monocular and foveal stimuli for accommodation as well as convergence are present. It has been shown that the refractive differential between binocular and monocular control of convergence is of the order of 0.25 D. in the cases which have been studied. In general, the indicated lead of accommodation is less under binocular than under monocular control. (Briefly, "lead of accommodation at the far point" may be described as the amount of negative power required to produce maximum visibility when negative relative accommodation is prevented.)

Criteria of Emmetropia.—The sensitometric correction of ametropia is that which produces maximal visibility without the exercise of relative accommodation. Thus it is absolute in this respect so far as any physical constant can be so considered from a physiologic viewpoint. On the other hand, the usual static correction is that which yields an arbitrary degree of visual acuity. Thus it is empiric in this respect. In addition, the usual methods of measuring visual acuity are limited in precision. This presumably minor handicap is eliminated in the sensitometric procedures by the averaging of a sufficient number of quantitative measurements of visibility.

Dioptric Biases in Usual Subjective Tests.—The usual subjective procedures are clinically biased in the direction of plus power owing to the practice of determining the maximal amount of plus power which is acceptable without an appreciable loss in visual acuity.[1c] If it were not for this bias, the prescription

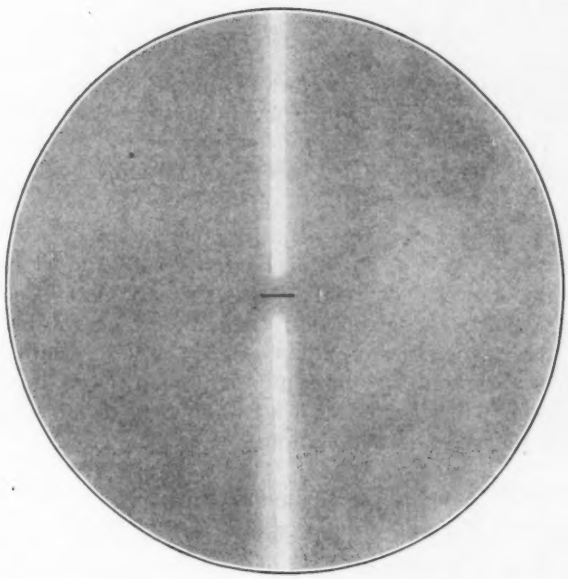

Fig. 2.—The convergence target and the biconcave test object, which are crucial components of this new sensitometric technic.

would be excessive in minus or deficient in plus power, and the not infrequent disparity between the usual subjective and objective methods would be greater than it is. Thus an apparent check between the usual subjective and objective tests appears to be due, in part, to empiric biases in the methods of testing. In general, it is probable that the plus power bias has been more or less indirectly introduced in static tests in order to obtain satisfactory corrections for near as well as for distant vision. If this assumption is correct, it follows that so-called static findings are not correctly designated as "distant corrections."

Negative Relative Accommodation.—The existence of a plus power bias in static tests favors the generally accepted theory that the normal adult eye is incapable of negative relative accommodation at distance. A priori this conclusion necessitates assuming that the adjustment of negative relative accommodation, which is available to the extent of several diopters in near vision, is absent entirely in distant vision. If this is a characteristic of the normal eye, it appears to be an

unfavorable one from the mechanical viewpoint of efficiency in making accommodative adjustments.

Ease of Seeing.—The normal adult nonpresbyopic eye is found to be myopic to the average extent of about 0.75 D. by the sensitometric method with binocular fixation and in the absence of relative accommodation. When the indicated myopia is corrected with lenses, prolonged and critical distant vision is found to be easier, as is revealed by a markedly lower rate of inovluntary blinking.[1a] If the eyes were actually emmetropic, rather than myopic at distance, it follows that the addition of —0.75 D. would constitute a stimulus for positive relative accommodation. Hence such a dioptric addition should augment rather than lower the rate of blinking during performance of a task demanding critical vision at distance.

Teleologic Considerations.—Since seeing is demanded at various distances and usually with frequent alterations in fixation, it seems improbable that the visual mechanisms would develop so that the state of ocular repose would be coincident with the extreme condition of distant vision.[1d] On the contrary, it seems more reasonable to assume that the "state of repose" would be coincident with some compromise position between the extremes of distant and near vision, particularly from the viewpoint of mechanical efficiency in shifting from long to short range and vice versa. The latter hypothesis is supported by the accommodation-convergence relationship determined for the normal subject by the sensitometric method.

Static Definition of Emmetropia.—Emmetropia is defined as "the normal state of the ocular refractive system in which infinity and the retina are conjugate with or without the exercise of relative negative accommodation." It is to be noted that although the static sensitometric examination indicates that the normal eye is myopic in the absence of stimuli for relative accommodation, it may become emmetropic when such stimuli are present. In certain cases involving prolonged, critical, distant vision it has been found advantageous to substitute negative lens power for negative relative accommodation.

DYNAMIC REFRACTION

Direct Dynamic Refraction.—The refraction in near vision is measured directly by the sensitometric method.[1e] At present this method is the only one which yields such information directly, as correction factors must be applied in other dynamic methods of refraction. Obviously such correction factors are the least appropriate in certain anomalous cases in which precise correction factors are most needed.

Control of Relative Accommodation.—The avoidance of this function by the sensitometric procedures appears to be as complete in near vision as it is in distant vision. Actually, the methods are identical at these extreme fixation distances. This is not a characteristic of other subjective methods.

Lag of Accommodation.—Since a blurred retinal image is assumed to constitute a stimulus for accommodation, it seems unlikely that a normal eye, capable of several diopters of relative accommodation, would remain definitely out of focus in critical near vision. If a deficiency in accommodation actually existed, its correction by relative accommodation should result in greater subjective fatigue and discomfort than appears to be experienced in prolonged near vision.

Effects of Prisms on Refraction and Visibility.—Exploratory sensitometric tests indicate that base-out prisms cause emmetropic eyes to become myopic, whereas base-in prisms produce a hyperopic condition.[1f] These refractive changes appear to

be approximately equal in magnitude at the usual reading distance. Obviously, equal and opposite refractive changes would not result if the accommodation lagged with respect to convergence. It has also been demonstrated with emmetropic subjects that base-out and base-in prisms of equal powers lower the visibility of a given test object by approximately equal amounts. Obviously, such a result would not be expected if the assumed lag of accommodation existed in reality. Tests made at a distance of 6 meters indicate that the so-called emmetropic subject obtains the highest degree of visibility with the addition (right eye) of a 4 D base-in prism. Since abduction results in a decrease in the refractive power of the eye, the attainment of maximal visibility at distance with base-in power is consistent with the static sensitometric finding of myopia with distant fixation.

Ease of Seeing.—The sensitometric examination reveals that emmetropic subjects obtain maximal visibility over a wide range of near vision without the addition of dioptric power. It has also been shown that the addition of either plus or minus power before the eyes of such subjects increases the effort involved in seeing, as indicated by a marked increase in the rate of involuntary blinking during the performance of a critical near vision task.[1a] If the accommodation lagged behind convergence in near vision, as has been postulated, the addition of plus power should increase the visibility of objects and make seeing easier. Neither of these results is obtained with additional plus power. In fact, the addition of plus power may slightly decrease visibility and markedly increase the rate of involuntary blinking.

Clinical Tests of the Sensitometric Method.—The corrections indicated directly by dynamic sensitometry have been shown to be appropriate in such diversified cases as those in which there are (*a*) normal or expected clinical data, (*b*) high dynamic retinoscopic and crossed cylinder findings, (*c*) low dynamic retinoscopic and crossed cylinder findings and (*d*) high degrees of exophoria or esophoria. Furthermore, the dynamic sensitometric corrections have been found to be satisfactory for both distant and near vision in those cases in which single ophthalmic corrections are possible.

Dynamic Definition of Emmetropia.—The normal states of adult nonpresbyopic eyes in which all points within the range of habitual near vision and the retinas are conjugate without the exercise of relative accommodation (and with the static addition), while more distant points may become conjugate by the exercise of negative relative accommodation.

Nela Park.

TUBEROUS SCLEROSIS

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Tuberous sclerosis, a disease entity first described by Bourneville in 1880 and often given his name, cannot really be considered by itself but must be looked on as a part of a group of diseases known as the phakomatoses. This name, derived from the Greek word φακός in its meaning of "birthmark," was suggested for the group because of the common characteristic of cutaneous lesions. There are now recognized in this classification four separate entities: tuberous sclerosis, or Bourneville's disease; neurofibromatosis, or von Recklinghausen's disease; angiomatosis, or von Hippel-Lindau disease, and, most recently added, the Sturge-Weber syndrome. All are characterized, as previously stated, by cutaneous lesions, which are respectively typical, and by an amazing variety of other lesions scattered throughout the body. The lesions, however, are predominantly important in the eye and make the syndromes commandingly significant to the ophthalmologist. But it is not the purpose of this paper to discourse academically on this disease group but merely to report a rather typical case of tuberous sclerosis which has recently come under my observation.

A 10 year old boy of Greek and Italian extraction was first seen in the clinic of the Manhattan Eye, Ear and Throat Hospital on March 11, 1942, having been referred by the school nurse.

The father and mother, about 36 years of age, were in good health. Both stated that their "eyes had always been good," and subsequent ophthalmoscopic examination revealed no abnormalities in the fundi of either. The patient was an only child; there had been a previous miscarriage. No history of any ocular trouble in the grandparents or other relatives could be elicited, but according to the parents a maternal uncle had died at 17 of "tumors of the heart" and the maternal grandmother had died at 61 of a "tumor on top of her heart which choked her" and which was spoken of as a "thyroma." Another maternal uncle, now in the Army, wrote that "the Army doctors had found something wrong in the back of one of his eyes."

The patient had seemed entirely normal at birth, but at 6 months he had attacks of generalized twitching and would stare straight ahead; these spells continued almost nightly. It was also noticed that he was cross-eyed. At 15 months he had a bad fall down the stairs. At 7 years he had an acute febrile illness which was diagnosed as encephalitis. He reputedly had a temperature of 107 F., was in a coma for four days and did not recognize persons for some time afterwards. The spells became less frequent after that, occurring only every ten to fifteen days, usually at night. According to the father's description of the attacks, there was rigidity as well as generalized twitchings and violent jerkings, more marked on the left side, twisting of the eyes and grinding of the teeth and usually unconsciousness.

He started school at the age of 6 years but stopped for some time after the so-called encephalitis, at the age of 7, and again for a year at the age of 9, on the advice of his teacher, supposedly because of nervousness. He had just returned to school in an open class, and his parents admitted that he did very poorly in his work and was not cooperative.

External examination revealed fairly typical adenoma sebaceum of the face, with a butterfly distribution across the bridge of the nose and cheeks (fig. 1). The eyes showed a convergent, nonalternating strabismus with poor central fixation of the left eye, but the extraocular movements were well performed. The scleras were white, the pupils were entirely normal and the tension seemed normal on finger palpation.

Read before the Section of Ophthalmology, New York Academy of Medicine, Dec. 21, 1942.

Uncorrected vision in the right eye was 20/20 —2 and in the left eye 20/400 eccentrically. The fields were difficult to take, but there was good light projection in both eyes. A roughly charted field for the good eye showed marked generalized constriction.

The slit lamp examination gave entirely negative results. The ophthalmoscopic examination, however, disclosed a striking picture. The media were entirely clear and the fundi well seen. Figures 2 and 3 show drawings of the fundi.[1] Below the disk and adjacent to it there was a large flat whitish area, and above the disk there was a smaller, similar area in the center of which there were a few tiny cysts. Temporal to this there was a fully developed, though small, cystic swelling. This is of interest because it supports the view that the flat whitish tumors often seen in this condition are simply precursors of the more typical elevated cystic tumors. The left eye showed a much more advanced picture. There were three tremendous cystic swellings, one on the disk, and one above and one below the macula. In spite of the size of these lesions, no hemorrhages or cystic buds floating free in the vitreous, as frequently reported, were seen.

Roentgenograms of the chest and sinuses were normal, the Wassermann reaction was negative and a roentgenogram of the skull was reported to show "three or four areas of

Fig. 1.—The patient, showing adenoma sebaceum of the face.

calcification in the brain substance, probably in the choroid plexus." General physical examination revealed nothing further except some patches of vitiligo, a few pigmented lesions and several small fibromas scattered over the body.

The patient was seen by a neurosurgeon, Dr. Thomas I. Hoen, who had an electroencephalogram made. The record was reported abnormal and compatible with a diagnosis of tuberous sclerosis.

The patient was last seen on December 9. He had been taking phenobarbital for five months, and his father reported that there had been no convulsions except several which had accompanied an undiagnosed acute febrile illness one month previously. He was, however, having considerable difficulty in school, and the parents often found him "very hard to handle," as they expressed it. There were a new small flat whitish lesion in the lower temporal quadrant of the right fundus and a possible slight increase in size of the old lesions, but otherwise no change in the ocular condition.

1. It should be explained that a mistake was made in the drawing reproduced in figure 2, so that it appears to be the left fundus, but it is really a mirror image of the right fundus and the lesions are all correct in their relation to the disk.

Fig. 2.—Drawing of the right fundus (owing to the artist's error this is a mirror image), showing flat whitish tumors and early mulberry-like tumors.

Fig. 3.—Drawing of the left fundus, showing advanced mulberry-like tumors.

This case is of interest because it presents the classic triad of Bourneville: mental deficiency, epilepsy and adenoma sebaceum, and, in addition, roentgenologic and electroencephalographic changes in the brain, as well as the striking ocular lesions.

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DISCUSSION

DR. THOMAS I. HOEN: Tuberous sclerosis has been known as a clinical entity since the middle of the last century, but cases are rare and it has been difficult to follow the patients throughout the course of the disease, so that the complete pathologic picture is not so well known as the clinical picture. It is known, however, that the abnormalities seen in the retina are a part of a general neoplastic tendency on the part of the patient. Many persons have thought the condition was limited to tissues of ectodermal origin, but this is not true, as rhabdomyoma of the heart and other forms of mesodermal tumor are frequently found. However, the picture is characterized by striking changes in the fundi, the skin and the central nervous system. The changes in the central nervous system are multiple enlargements, for example of glial tissue, often considered not to be true tumors, although I believe it is fairly generally recognized now that they are true tumors of glial origin. The cells of the tumors in the fundus and in the brain are typical of the cells of tumors of the astroglia group as classified by Cushing and Bailey. They have the typical fibrillary structure, many tube-feet attached to blood vessels are seen and they frequently undergo cystic degeneration, which is also a characteristic of astrocytomas in general.

I saw the boy described by Dr. Constantine, and we had an electroencephalogram made, which I shall not describe except to say that there were abnormal waves of average voltage and a slow rhythm, which are characteristic of neoplasms in general. The fact that the slow waves could be picked up over various parts of the cortex strengthened the feeling that the boy had numerous tumors in the brain. A pneumoventriculogram could have been made. The characteristic picture, instead of the pretty butterfly picture, shows irregularities and distortion of the ventricular contour. It is common to find tumors of the choroid plexus; they are waxlike nodules on the choroid called "candle drippings." The calcification in the choroid shown by the roentgenogram might have been in a tumor because such tumors often undergo calcification.

The clinical and the pathologic picture are well known, but there is no known method of treatment other than symptomatic handling. This patient had seizures, and he was known to be mentally deficient. He was given anticonvulsant drugs, which stopped the seizures; nothing could be done to improve his mental status or prevent loss of vision from the tumors in the fundi. We hope we shall be able to follow this case to its ultimate conclusion, which is not far in the future. Some authors have recommended decompressive operations in such cases, but I do not believe that they are satisfactory. If signs of intracranial hypertension are present, the decompression will prolong life somewhat, but not very happily.

In conclusion, I would say that this condition, while rare, is more common in certain places than in others. It was identified first in continental Europe and is more common there. It is rare among Anglo-Saxons, occurs in Negroes and occurs rather frequently in the Japanese.

DR. HARRY MCGRATH: About three years ago I had the opportunity in a mental hospital of observing a case of tuberous sclerosis with the typical syndrome of adenoma sebaceum, convulsions and mental deficiency. There was a smooth white mass on and above the disk in the left eye. It was not of the mulberry type described by Dr. Constantine. There was no other lesion in either fundus.

In the course of four months there was no change in the shape and size of the tumor. At the end of that time the woman suffered fracture of the skull in a

fall, and she died shortly afterward. The eye became available for microscopic study. This case, with the pathologic study, was reported in a neurologic journal but has not been mentioned, to my knowledge, in the ophthalmologic literature, although I understand it was only the second case in the United States in which a microscopic study of such a tumor was made.

Grossly the tumor was cystic. It had its origin in the nerve fiber layer and involved the rest of the retinal layers only by pressure. The cells were astrocytes with processes embracing the vessels. The pathologist who examined the sections was of the impression that it was a glial tumor of low growth possibilities and not a congenital malformation. The sections are available for study.

REOPERATIONS FOR GLAUCOMA

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The return of hypertension after an apparently faultless operation for glaucoma is a bête noire of the ophthalmic surgeon. It cannot be predicted or forestalled. It must simply be accepted as one of the hazards of glaucoma to be dealt with as well as possible.

The literature is replete with indications and contraindications for primary operations, but there is little general agreement as to the relative efficacy and value of the operations themselves. This is undoubtedly due to a lack of understanding of the etiology and pathology of glaucoma. Each operation is designed to relieve only the hypertension, by removing the obvious barriers to filtration or by providing a vicarious pathway. The underlying disease is not cured by the removal of one of its symptoms.

In spite of innovations and modifications of operations for glaucoma, little factual or practical progress has been made within the past twenty-five years. Statistical reports of the different operations reflect a wide variety of choice on the part of individual operators, with no universal lessening of the percentage of failures.

If one cannot feel satisfied with the present status of initial operations for glaucoma, one must feel still more inadequate when confronted with the necessity of a secondary operation.

After the first operation the eye is no longer the same. Its anatomy has obviously been altered. The physiology of secretion and drainage, which depends so much on the integrity of the nervous and vascular systems, has suffered a profound shock. The patient himself cannot be ignored, for in its broader aspects the psychosomatic interrelationship plays its part in the production of glaucoma and certainly in the behavior of the glaucoma in the postoperative state.

If careful, prolonged study of the anatomic structure and reactions of the eye should precede a primary operation, it must be accepted as equally important, perhaps more so, when a secondary operation is contemplated. After a secondary operation it becomes a matter of dealing specifically with the complications that have arisen or of trying to reduce the tension in some other way.

The most optimistic reports of antiglaucoma operations list 10 per cent failures, whether or not the after-treatment involves medication. The period before the return of hypertension varies from a few weeks to many months. Schoenberg's case in which the hypertension returned after eighteen years can hardly be listed as an instance of operative failure!

Errors in judgment naturally increase the number of failures, but even when the operation is well chosen and technically sound, failures occur. Then the cases are analyzed, and the student is presented with an overwhelming number of reasons to account for the failures. Most of the evidence comes from the pathologic laboratory. Lately, with the more universal use of the gonioscope, the young ophthalmologist has learned to look for the reasons for failure in the living eye. In this search, too, the literature will help him, with its many references.

Read before the New York Academy of Medicine, Section of Ophthalmology, Nov. 16, 1942.

But there the matter seems to stand. The patient still has glaucoma. Medication, then operation and then postoperative treatment have failed. What should be done? The literature is strangely quiet on this question, and the operator has the awful feeling that he is now entirely on his own, God help him!

The subject of reoperations seems important enough to warrant discussion. Every ophthalmologist of experience has been faced with the problem and expects to have it recur again and again, but human nature is reluctant to publish its failures, and the results of experience in this matter have seldom been committed to the printed page. In view of the paucity of material, the approach to the subject is not easy. What little can be said, therefore, must be limited to a few personal experiences and conclusions and the results of conversations with others.

The tension is hardly expected to become normal immediately after operation. As soon as the eye is opened and the pressure drops, there is edema of the ciliary body, and even though an attempt is made to "stabilize" the pressure by the injection of air or saline solution and the use of water-tight sutures, a normal condition does not exist. The eye must be allowed a decent interval to adjust itself to its altered anatomy and physiology. A slight postoperative rise in tension should certainly cause no alarm, and no one would be importunate enough to rush into a secondary operation.

But when the need does arise, a pathologic tension must be combated by whatever measures seem appropriate.

The following case serves to illustrate the unusual amount of operative intervention that sometimes seems justified. Moral support was obtained through repeated consultations with other ophthalmologists, who concurred in the proceedings.

Mrs. F. D., a white woman aged 56, had a Lagrange sclerectomy with peripheral iridectomy for chronic noncongestive glaucoma. The operation appeared satisfactory, but twenty-four hours later the anterior chamber was completely filled with blood. A rise in tension was feared, but the Schiøtz tonometer readings were not above 23 mm. of mercury for ten days. Then the eye became stony hard. Irrigation of the anterior chamber through a large corneal keratome incision washed out most of the blood, and clot forceps removed the remaining clot.

The eye quieted down almost immediately, and the tension stayed around 19 mm. of mercury for nine days. Then development of a low grade iritis with posterior synechias necessitated intensive treatment with mydriatics, during which the tension went up to a new high. It was brought down by paracentesis repeated twice a day for a week. The synechias were still present.

It was felt that no real progress was being made, and so the eye was opened again at the site of the original Lagrange operation. The sclerectomy incision was still patent, and through it the iris adhesions were pulled free. The peripheral iridectomy was made complete and the temporal pillar incarcerated in the sclera.

The eye finally quieted down, and later ophthalmoscopic examination disclosed the remains of a large hemorrhage into the vitreous. In view of this final discovery, it is doubtful whether the eye had much of a chance after the blood filled up both the anterior and the posterior segment.

It is not pleasant to report such a failure, but to do so will serve a purpose. It was not known at the time what else to do, nor is it easier in retrospect to decide whether something else should have been done.

Undue hemorrhage is always a matter of concern. When it occurs in the anterior chamber and produces an inordinate rise in tension it can be dealt with in the manner described. In lesser amounts it may respond to medication.

When there is a posterior nonexpulsive subchoroidal hemorrhage, the blood accumulates rapidly and the eye becomes hard and painful. The anterior chamber is usually shallow. According to laboratory reports this type of hemorrhage occurs most commonly after trephining or after the Lagrange type of sclerectoiridectomy.

probably because the prolonged softness of the eye permits a rupture of the diseased blood vessels due to removal of the supporting intraocular pressure. Immediate posterior sclerotomy was effective in 2 cases at the New York Eye and Ear Infirmary in removing the blood and saving the eye. The Graefe knife was plunged in, twisted and withdrawn. Other operators report using a trephine operation or dissection to remove a piece of sclera, exposing the choroid. Then multiple diathermy punctures released the fluid. Incidentally, this same procedure (posterior sclerotomy) has been used as an adjunct to the main operation, to reduce the tension posteriorly before the anterior segment was opened.

Except when there is early hemorrhage, the eye operated on deserves a rest. Sedatives, medication applied locally and massage will usually control the tension during the immediate postoperative period. Later, even when the tension is just above normal and persistently refuses to come down, an extensive reoperation need not always be considered. In several cases at the Infirmary a corneal paracentesis or anterior sclerotomy with a keratome has apparently given just the necessary amount of easement to allow the eye to take care of itself.

If a primary operation is necessary, a reoperation is just as much needed when there is a mechanical failure of the first.

There are two main reasons for the failure of an external fistulizing operation: blocking of the filtration wound on the inner aspect of the eye and fibrosis of the overlying tissue.

The filtration wound is blocked most commonly by ciliary processes and iris tissue, sometimes by lens capsule or other debris and sometimes by the products of inflammation. The surprising thing is that so few eyes appear perfectly normal in the area operated on, as shown by gonioscopic studies.

The presence of uveal tissue in the wound is not necessarily an impediment to filtration (as a matter of fact, many ophthalmologists believe that it is a requisite to good drainage), but when it blocks the wound it vitiates the operation. Often when a knuckle of iris fills the opening, drainage can be reestablished by excision of a small piece.

For the past two years, in every case of operative failure in the Knighton clinic at the New York Eye and Ear Infirmary I have examined the patient personally in an attempt to discover the reason for the failure and in the hope that some remedy simpler than another extensive operation could be devised. In perhaps a dozen cases, about equally divided between trephinings and sclerectomies, the original conjunctival flap was redissected down to the filtration opening, and if any plug was found in the hole it was removed. The results were almost 100 per cent successful as far as the scleral drainage was concerned. The subconjunctival drainage was another matter. It has long been known that the prognosis is poorer after secondary operations, especially when dissection is made through healed tissue.

Even in primary operations, special emphasis has been placed on the necessity for dissecting the conjunctival flap close to the sclera under Tenon's capsule. This is supposed to allow better absorption of the aqueous, in the looser subconjunctival tissue that lies over the capsule. In spite of the most meticulous care in such dissection, many fistulizing operations fail because of fibrosis of the conjunctival flap. It was surprising to note in a number of eyes in which the incision was reopened, that the trephined hole or the sclerectomy opening was still patent. In each case, however, scar tissue was encountered at the exit of the filtration opening.

The reoperation of course only made matters worse by causing more scar formation.

The injection of air or saline solution at the primary operation may or may not prevent too firm an adhesion of the conjunctiva.

Injection of air is reported in 1 instance to have reopened the subconjunctival spaces enough to obviate the necessity for further operation. My experience in 1 case was not successful.

When the conjunctival wound is opened with a spatula, the tension stays down from two to five days before the wound is sealed off again. This procedure has been performed in several cases as an office procedure to carry the patient over until a secondary operation could be performed, but there can be no hope of permanent success from repeated traumatism of this sort.

In 1 case a horsehair seton was tried in the area operated on but with no better results. Its use seemed to delay the cicatrization of the conjunctiva a little longer, but that was all that was accomplished.

The patient was a white man 60 years old with chronic congestive glaucoma. A Lagrange sclerectomy and complete iridectomy failed to relieve the hypertension, although redissection of the conjunctiva disclosed a scleral opening that was draining nicely. There was diffuse scarring of the conjunctival flap. A horsehair seton was inserted under the conjunctiva at the sides of the scleral opening and run laterally on each side, as close to the sclera as possible and under conjunctiva that had not been dissected previously. The free ends were buried. It was hoped that the seton would keep a path open to the fresh conjunctiva, but the experiment failed miserably. In eyes not previously operated on, the seton has given better results—even in 2 cases of absolute glaucoma.

The question of repeating the operation is always present. Should the same operation be tried again, or should one choose another type on the assumption that the first one failed because it was the wrong choice?

The answer is not easy. When an apparent choice was offered, the operator undoubtedly selected the original operation because it was the one in which he had the greatest skill and the most confidence. If he was asked to perform a less familiar secondary operation on the simple grounds that the first choice had failed, he would be justified in asking why. On the basis of the operator's ability alone, the eye would probably be safer under a repetition of the procedure in which he was most expert. And if there were technical errors or complications that could be avoided, a second performance would seem justified. But if the original operation was performed to the best of the operator's ability and there were no complications, a failure would mean that the operation was not good enough. It is difficult to know in such cases whether the fault lies with the operator, with the type of operation or with the reaction of the eye in question. But the evidence of failure seems to indicate some kind of error and the necessity for a change. A good operator has more than one string to his bow and is ready to try another when the first gives poor results.

Of the external fistulizing operations, trephining is the first choice, followed by sclerectomy and iridencleisis. The same order of preference seems to obtain in the matter of secondary operations, although some operators prefer a sclerectomy when there is an exacerbation of the glaucoma itself, in contradistinction to inadequate drainage from the incision made in first operation.

When there are signs of irritation with the return of hypertension, cyclodialysis should not be considered as a secondary operation, but a complete iridectomy should be included in the operation selected. Cyclodialysis is often satisfactory as a secondary procedure, especially in aphakic eyes, but it should not be attempted unless the angle is partially open.

There has been a recent swing in favor of iris inclusion operations either alone or in combination with sclerectomy, even as reoperations. My experience, however, has indicated that the best results are obtained when the iris tissue is healthy and not inflamed. In glaucoma of long standing the results have been poor.

The choice of a site for a secondary operation deserves careful study. Transillumination of the eye often prevents later embarrassment by disclosing the atrophic parts of the iris to be avoided, and the scleral scatter outlines the ciliary body. Gonioscopic examination is of course indispensable.

The presence of trabecular or scleral iris adhesions often moves the angle so far forward that trephining into the anterior chamber is impossible. Trephining through the iris into the posterior chamber may succeed, but there is always the chance of incarcerating ciliary processes. If the operation is not spoiled mechanically, it may fail later as a result of a low grade uveitis from irritation of the ciliary body. A Lagrange type of iridosclerectomy in the same circumstances would be impossible.

I have obtained some success by making a horizontal scleral incision with a scalpel just behind the limbus and entering the eye as in iridectomy *ab externo*. The iris is freed from the anterior scleral lip enough to permit a sclerectomy with scissors. Then a small piece of iris is excised. A conjunctival flap has previously been laid down in the usual manner. In 2 recent instances a small amount of fluid vitreous escaped, but except for a continued low tension, about 12 mm. of mercury, there have been no untoward results.

Postoperative hypotony may reach serious proportions and has been reported corrected by the bringing down of a conjunctival flap over an area of denuded cornea. The subsequent fibrosis is usually sufficient to elevate the tension. Good results following the application of a thermophore to the filtering bleb have been reported.

A shallow anterior chamber is difficult to handle, and it is not always made deeper by posterior sclerotomy, as some advocates claim. My attempts to open the angle by injection of the anterior chamber with air or saline solution have met with indifferent success. Preference is still held for the *ab externo* approach. In several private cases a swollen lens was removed with good effect on the tension and a deepening of the anterior chamber. In one of these the extraction was carried out through an old glaucoma filtration wound, destroying its effect, but the tension remained normal. The same procedure in a clinic case caused a postoperative return of the hypertension, which had to be controlled by another operation. The lens should be removed in capsule whenever possible to avoid later blocking by lens or capsular debris.

In spite of all precautions and attempts to prevent absolute glaucoma, in many eyes this condition develops. For it, of course, the perfect reoperation is enucleation, but when the patient protests, the surgeon must temporize. Simple posterior sclerotomy will often relieve the pain, although it seldom effects a permanent reduction in tension. Diathermy puncture through a posterior scleral trephine opening has been reported to give the same result. Opticociliary neurectomy has been effective in relieving the pain, and injection of alcohol into the ganglion has produced the same result. Perhaps other measures can be justified on the basis of expediency.

These are a few of the secondary attempts that have been made to rectify an initial operative failure. Many other methods have been tried with varying success but have not been mentioned because of lack of familiarity with them.

SUMMARY

Reoperations are necessary in the treatment of glaucoma because primary operations are not always successful.

Except when obvious faults of the primary operation can be corrected, a reoperation has less chance of succeeding because of physiologic changes, mechanical difficulties, fibrosis or other unknown factors.

Any operation is a decided shock to the eye and its functioning; the postoperative state may differ widely from the preoperative state and requires careful restudy before further intervention is considered.

The subject of reoperations deserves more discussion than it has received.

121 East Sixty-First Street.

FIXATIONAL CORNEAL LIGHT REFLEXES AS AN AID IN BINOCULAR INVESTIGATION

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MEDICAL CORPS, ARMY OF THE UNITED STATES

The estimation of binocular imbalance based on objective examination of the eyes has been found unsatisfactory because inspection of the eyes in a routine examination of the muscles is often difficult. The role of the corneal light reflex as an aid in clinical practice has not been sufficiently explored, and my aim in this paper will therefore be to show how this reflex can be utilized clinically to complement the familiar cover test.

First, as to the meaning of the fixational corneal light reflex: For practical purposes it may be regarded as the constant position of the light reflex on the cornea when the eye is made to fix a light and when the observer's eye is behind the testing light. It may not be on the center of the cornea (angle gamma), even though the macula assumes fixation, but it represents the normal anatomic position of the light reflex for that fixing eye and is to be distinguished from the more peripheral, or deviational, corneal reflex seen in a squinting eye when the fellow eye is fixing.

With an ordinary flashlight one notes whether fixation is as satisfactory binocularly as monocularly. The examiner keeps his eye directly behind the test light and makes a mental note of the position of the light reflex on both corneas. He then covers each eye alternately and compares the binocular reflexes with reflexes obtained by monocular fixation. If the corneal light reflexes assume the same positions binocularly as they do on monocular fixation, deviation is absent for that testing distance. Displacement of the light reflex from the fixation position in one eye when both eyes are tested indicates deviation in that eye. If the reflexes appear centered on binocular stimulation but displacement occurs in one eye on attempted monocular fixation by the alternate cover test, false macula is suggested.

If one regards a fixational corneal light reflex as defined as indicating normal macular projection, displacement of the light reflex from the fixing position would indicate registration of the image on an extrafoveal portion of the retina. If a light is directed on the cornea from the nasal side so as to produce a corneal reflex medially, registration of the image on the retina will be temporal. One may simplify matters by stating that the position of the corneal light reflex in relation to the fixing position indicates the direction in which the image is being projected (fig. 1). If the reflex is nasal to the fixing position, projection of the image is also nasal.

In terms of binocular imbalance, the corneal light reflex in divergent squint would be nasal in the deviating eye, or in a direction opposite to the deviation, and diplopia, if present, would be nasal, or crossed, to correspond to the nasal displacement of the reflex. When the reflex is displaced upward and nasally while the other eye fixes (fixational reflex), there is a downward as well as an

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outward displacement of the eyeball (hypoexotropia), an inferotemporal registration and, again, an image projection (upward and nasalward) corresponding to the displaced position of the corneal light reflex in relation to the fixing position.

To appreciate more fully the meaning of a corneal light reflex one should bear in mind the following principles:

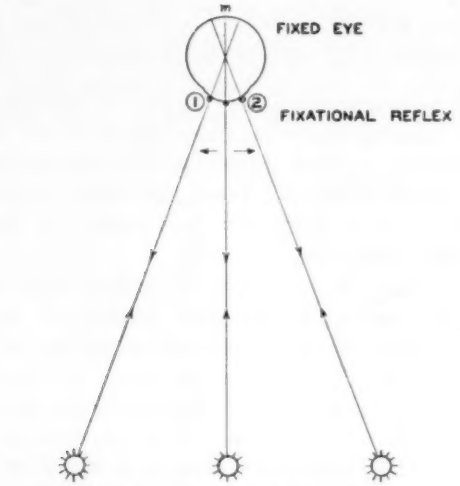

Fig. 1.—Position of the corneal light reflex indicates image projection.

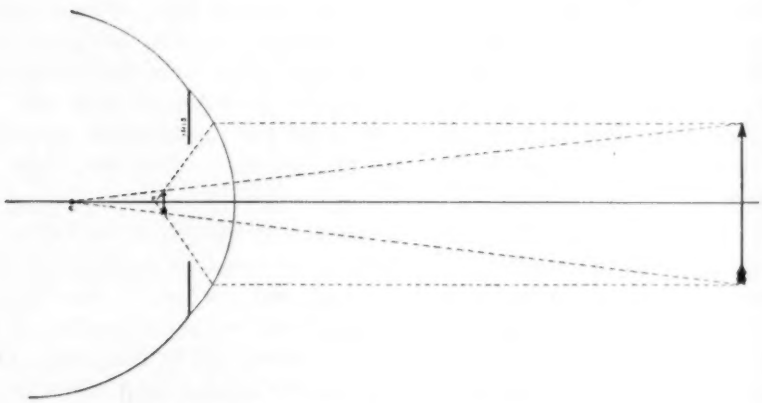

Fig. 2.—Relative size and position of image with convex mirror.

A. The apical portion of the cornea is a convex spherical mirror and therefore the first, or anterior corneal, reflex has all the characteristics of an image produced by such a mirror (fig. 2).

B. The corneal light reflex is diminished in size, erect and virtual.

C. Any ray incident through the center of curvature will return along the same path. An incident ray parallel to the principal axis will be reflected as coming from the focal point, which is located one half of the radius behind the surface.

D. Owing to the relatively small radius of the cornea (about 8 mm.), the image plane for any test light distance will practically coincide with the focal plane 4 mm. behind the surface. Even if the test light is 333 mm. from the cornea, the difference in location is only 0.05 mm.

E. The anterior, or bright, Purkinje corneal reflex, therefore, strictly speaking is not a superficial reflex, but is retrocorneal, or 3 to 4 mm. behind the surface.

F. As a light is moved parallel to the facial plane of the patient the corneal light reflex moves in the same direction, although to a much reduced extent.

G. The position of the corneal light reflex depends on the position of the test light relative to the principal axis and the eye of the observer. The distance from the test light to the cornea is of no practical influence, for the reasons mentioned.

It is important for purposes of accuracy to adopt a uniform means of studying corneal light reflexes. The position of the examiner's eye behind the light source is found to be most reliable and uniform.

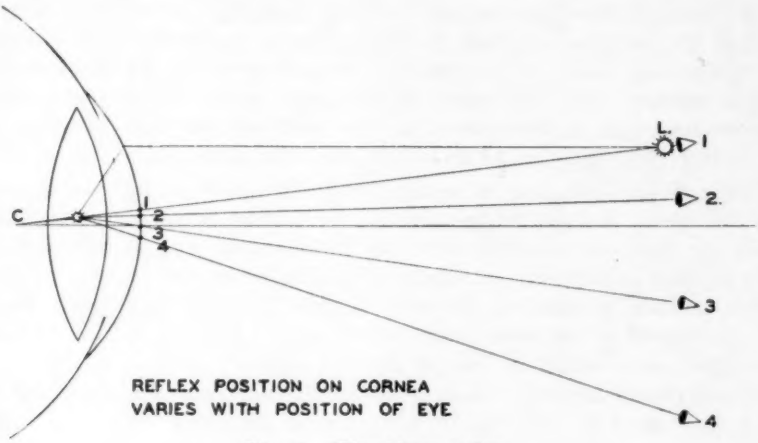

Fig. 3.—Hirschberg reflex.

If one assumes that fixational corneal light reflexes indicate an absence of deviation, one can reason that any artificial means of centering a peripheral corneal light reflex, as in squint, might likewise serve as a basis for measuring ocular deviation. In the case of the synoptophore, the carriers are rotated until they are in direct line with the corresponding fixation positions on the corneas, and readings are made. This is a far more satisfactory method than the crude Hirschberg test, based on translating roughly the position of the corneal light reflex in a squinting eye in terms of millimeter displacement from the fixing position.

The method involving the peripheral corneal light reflex (Hirschberg, fig. 3) is unsatisfactory for the measurement of squint because:

1. It is based on a rigid value (8 mm.) for the corneal curvature, which must vary in different persons.

2. It is difficult to control the patient sufficiently to measure the position of the light reflex accurately. Moreover, the position of the reflex changes with movement of the examiner's eye.

3. The amount of millimeter displacement can be estimated only roughly and is never found to be uniform when measured by different examiners. Moreover,

a displacement of only 1 mm. means 7 degrees, and one is in no position to make fractional readings.

4. The method does not take into account the center of rotation of the eyeball in addition to the center of curvature of the cornea.

5. The corneal curve is not a geometric curve, because flattening toward the limbus takes place.

6. A corneal light reflex seen peripherally is distorted, whereas one seen at the center of the cornea is sharp.

Artificial restoration of fixational corneal light reflexes for measurement of ocular deviation may be obtained as follows:

1. By ordinary flashlight. In convergent squint, the light reflex on the nonfixing eye will be displaced temporally from the corneal fixation point. As the test light is gradually moved along a rule closer to the eyes, the light reflexes reach the corneal fixation points, and on the basis of the patient's interpupillary distance and the distance of the light from the eyes (near point of convergence), one can calculate readily the amount of squint. In actual practice, however, this method of testing convergent squint is found wanting. One must be careful to stop just at the position at which the lights appear centered, or an added convergence impulse may upset one's reading; measurement of the interpupillary distance in a patient with convergent squint may prove difficult, and, finally, the reading obtained can be interpreted as the deviation for that distance only and not for 20 feet (610 cm.) or 13 inch (33 cm.) test distances.

2. By means of a partition, or septum, and individual test lights at a set distance between the eyes. A light on one side of the septum is set in front of the fixing eye, while the light on the other side, for the nonfixing eye, is moved laterally or vertically or both until it, too, produces a light reflex on the corneal fixation point. When this position is reached, alternate fixation to light stimulation should produce no movement of the eyes unless amblyopia is present in the nonfixing eye. The examiner's eye should be behind the test light.

A septum placed between the eyes serves the purpose of a cover but with the difference that the two eyes can be observed at the same time. In orthophoria the two eyes project to a common point on the cover test. When the cover is moved into a septal, or between the eyes, position, fixation by one eye on a corresponding light placed close to the septal wall will make the fellow eye converge in the direction of the unseen light in the case of orthophoria. In exophoria or exotropia the nonfixing eye turns out, and the test light is displaced laterally to reach the corresponding corneal fixation point. In esophoria or esotropia the light reflex on the deviating eye is displaced laterally to the fixation point, and supplementary base-out prism is required to center the light reflex in the deviating eye.

Use of the septum arrangement is of distinct advantage not only as a quantitative cover test but as a comprehensive means of comparing objective and subjective responses, notably in cases of abnormal retinal correspondence. When the images appear to touch or to correspond at the angle of deviation, it speaks for so-called normal retinal correspondence because the subjective, or image, angle corresponds with the objective, or deviation, angle.

3. By means of the synoptophore or the stereoscope. The synoptophore and the stereoscope are essentially devices which utilize the principle of the septum, or independent stimulation to each eye. They also incorporate plus lenses to relax accommodation and convergence, thus bringing parallelism of the visual axes to

within a comprehensive range. Whereas without the septum the eyes normally tend to converge on opposite sides of the septum, the incorporation of plus viewing lenses tends to shift the visual axes outward.

The basis for centering of the corneal light reflexes with the synoptophore appears simple. By rotation of each carrier around a pivotal point the mirrors are so rotated as to bring the corneal light reflexes to their respective fixing positions at the angle of deviation.

With the Brewster stereoscope [1] a critical corneal light reflex can likewise be obtained by use of bright transparent targets that do not cover too large retinal fields. As these targets are placed in the calculated primary positions for different accommodations one may readily note whether centering of the corneal light reflexes is maintained. The angle through which such targets are displaced medially or laterally relative to the primary positions in order to strike the centers, or fixation

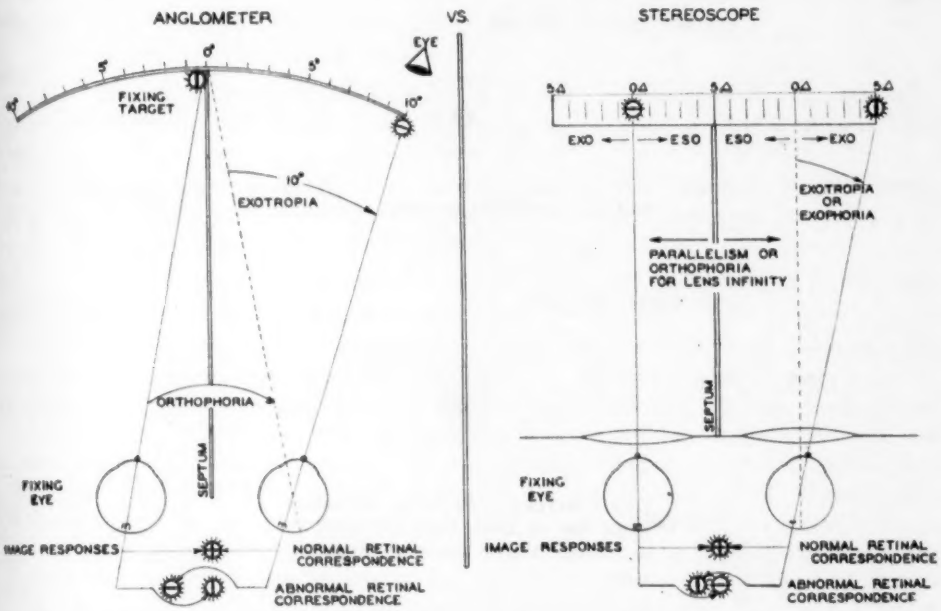

Fig. 4.—Comparison of anglometer and stereoscope, showing positions of targets in relation to ocular deviation, fixational corneal light reflexes and image responses.

points, of both corneas serves as a measure of the amount of ocular deviation for that testing distance.

The anglometer,[2] also incorporating a septum, provides for studies of the corneal light reflexes at but one accommodative range (3 D.), or at a fixed distance of 13 inches (33 cm.); its flexibility lies in its permitting one to study the positions and visual responses of the eyes in the different motor fields. My stereoscope permits one to study the corneal light reflexes in a single motor field or with the eyes-front gaze, but its flexibility lies in its enabling one to study the corneal light reflexes at different viewing distances or accommodative ranges.

1. Krimsky, E.: The Stereoscope in Theory and Practice, Brit. J. Ophth. **21**:161-197 (April) 1937; Some Newer Developments in Precision Type Stereoscopes, Arch. Ophth. **19**: 394-402 (March) 1938; Modifications of the Brewster Stereoscope for Clinical Requirements, ibid. **26**:808-815 (Nov.) 1941.

2. Krimsky, E.: The Cardinal Anglometer, Arch. Ophth. **26**:670-674 (Oct.) 1941.

4. With the aid of a prism—the prism reflex test. In the routine study of ocular deviation a prism is used to nullify ocular motion on alternate covering. The role of the variations in the position of the corneal light reflex as a result of use of the prism has been largely overlooked, possibly for the reason that test targets set at a distance of 20 feet are too faint to yield visible corneal light reflexes. In studying the effects of a prism with a test light at a 13 inch reading distance I found that, whereas a ray of light traversing such a prism was refracted toward the base, reflection of the light on the cornea was toward the apex. I found, moreover, that a prism sufficient to stop motion of the eyes on alternate covering also had the fortunate property of centering the corneal light reflexes. This interesting phenomenon, to my knowledge, has not been reported previously in the medical literature. So here there is additional proof that whatever method one employs the primary aim in measuring latent or manifest squint is, first, to

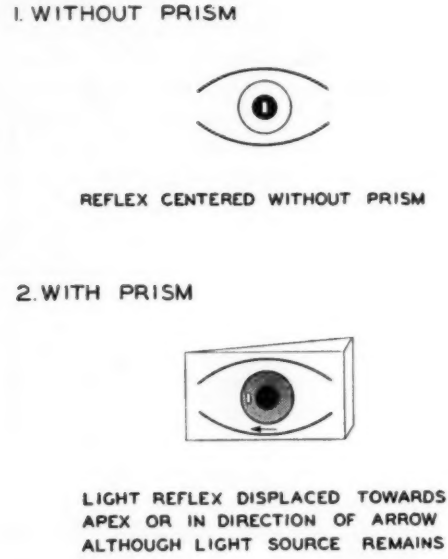
1. WITHOUT PRISM
REFLEX CENTERED WITHOUT PRISM
2. WITH PRISM
LIGHT REFLEX DISPLACED TOWARDS APEX OR IN DIRECTION OF ARROW ALTHOUGH LIGHT SOURCE REMAINS FIXED.

Fig. 5.—Effect of prism on glass eye.

produce artificial restoration of the corneal light reflex from a peripheral to a fixing position, and with prisms to ascertain the strength required to center these reflexes artificially.

One can observe easily the change in position of the corneal light reflex with gradual increase in prism by experimenting with a glass eye and a rotary prism. The light is directed to the center of the pupil, and both the observer's eye and the test light are kept in line in a steady position. As the prism is rotated in either direction, it will be noted that the light reflex moves in the direction of the apex of the prism and that a slight increase in strength of the prism will produce appreciable displacement of the light reflex. The eye itself will also be seen to move toward the apex of the prism, but such gross change in the apparent position of the eyeball is distinct from that of the corneal light reflex because it is on a different image plane. One can simulate deviation by turning the glass eye in or out in relation to the test light, which remains in the same position. When the eye is turned out, the light reflex is seen to move in from the fixation

or the central position. To bring the light reflex back to the fixation position, one merely sets the prism with the apex in the direction of the center of the pupil. Comparison of the setting of the prism to the direction which an arrow must take to reach the center of the pupil simplifies the matter.

The prism reflex test simplifies the measurement of ocular deviation. The novice confronted with a patient with squint may be at a loss as to how to place his prism in order to neutralize the deviation. If he thinks in terms of fixational corneal light reflexes, he merely turns the prism first one way and then the other and notes the direction and amount of prism (using a rotary prism) required to bring the light reflex in the deviated eye to a central position. In esotropia the eye is turned in and the light reflex is displaced temporally, and a base-out prism centers the corneal light reflexes accordingly. If the eye turns upward as well as inward, the light reflex in the deviated eye is displaced downward as well as outward and both a vertical (base-down) and a base-out prism bring the light to the center of the cornea in that eye.

Certain precautions should be observed:

A. This test is most satisfactory for a 13 inch test range. For greater distance, a stronger light source must be employed to yield a bright corneal light reflex.

B. A rotary prism is the most satisfactory instrument for the test. A phorometer is generally unsatisfactory, because the framework covers the eyes in such a way as to make careful inspection of the reflexes difficult.

C. As a rule the prism is placed over the fixing eye, and changes in the position of the corneal light reflex are observed in the deviating eye.

If in a case of manifest squint, the corneal reflex is displaced only laterally (horizontal squint), this simplifies prism testing, in that a rotary prism alone is sufficient to produce centering of the corneal light reflexes. If the displacement is oblique, in other words the squint is vertical as well as horizontal, one first corrects the vertical component by using a vertical prism, always remembering that the light reflex will shift in the direction of the apex of the prism. Specifically, if the reflex in the deviated eye is higher (hypotropia), one first levels the reflexes by placing a prism with the apex down. As a rule the vertical component requires much less prism than the horizontal component, and when this correction is disposed of, one is in a position to neutralize the horizontal component with a rotary prism placed in front of the vertical prism. The rotary prism is rotated so that the light reflex moves in the direction of the apex of the prism. If the reflex in the deviated eye is displaced medially (exotropia), one rotates the prism base in, or apex outward. After having obtained what appears to be centering of the light reflexes in both corneas, one supplements the procedure with the familiar cover test and ascertains whether ocular motion has been stopped and whether the light reflexes remain steady on alternate covering. It often happens that the results of this rapid preliminary prism reflex test conform closely with those of the cover test. In other instances, just a little more turning of the prism in either direction is sufficient to stop the movement. At any rate, one sees the eyes and their reflexes much more clearly than one does by relying on ocular movement alone.

The prism reflex test does not replace the familiar cover test but simplifies it by permitting rapid inspection of the restoration of the corneal light reflexes to their fixation positions. The cover test alone is not infallible, for if one has to rely entirely on the gross appearance of the eyes one may be at a loss to tell exactly when movement has stopped. It requires a critical eye to judge cessation

of fine ocular movements, and the prism reflex test proves helpful as a rapid confirmatory test. Also, the cover test is deceptive in certain types of manifest squint associated with false macula, in which there is no movement of the eyes on alternate covering. In this case the prism reflex test offers a satisfactory estimate of the amount of squint.

As a rule, the examiner selects one eye for placement of the prism. I usually place the prism before the right eye. However, in the case of severe squint, especially with amblyopia, one sets the prism before the normal, or fixing, eye and then by rotating it in the direction of neutralization observes the corneal

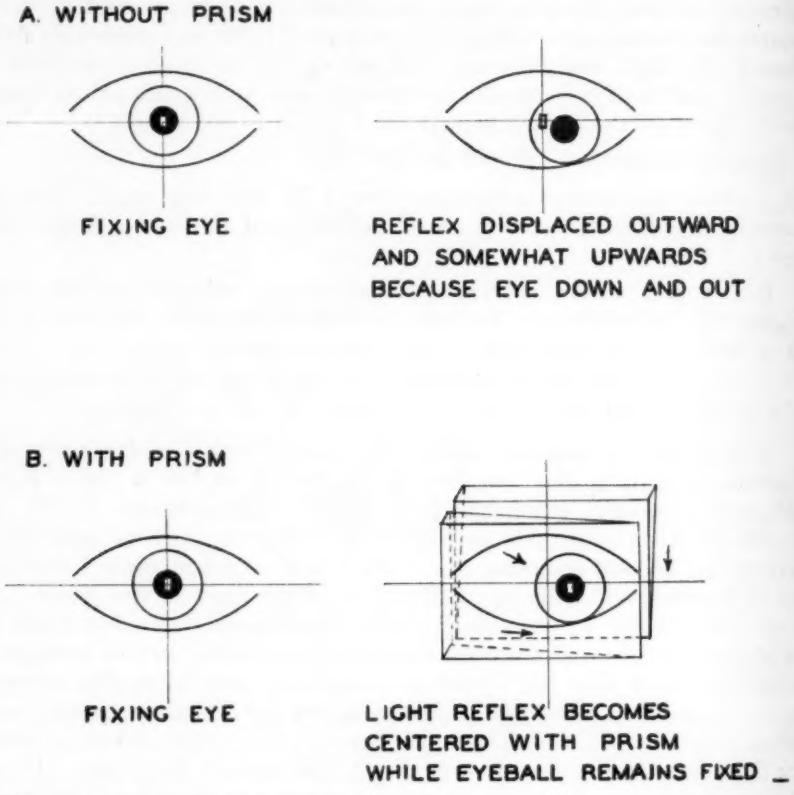

Fig. 6.—Hypoexotropia, showing restoration of fixational corneal light reflexes by prisms.

reflex becoming gradually centered in the otherwise deviating eye because the fixing eye moves in the direction of the apex of the prism. For this test I prefer the simple rotary prism to the bulky phorometer, because it covers one eye rather than both, it is free from obstructing framework, it can be used on either eye and it can be used for the study of vergence as well as deviational response. It is also preferable to individual square prisms.

For excessive deviation no prism is reliable, and the angle method as embodied in the cardinal anglometer is the method of choice. The anglometer enables one to study deviations in all selected positions of gaze and has certain controlled features which render the readings reliable. Moreover, it is based on physiologic and optical principles which make it as sound as is feasible for clinical requirements.

CLINICAL APPLICATIONS OF THE CORNEAL LIGHT REFLEX TEST

1. *Pseudosquint, Angle Gamma; Epicanthus.*—In studying the corneal light reflexes, one first determines their position on each eye with monocular fixation. Whether the reflex is found centered or somewhat off center (angle gamma), for practical purposes one calls its location the corneal fixation point because it corresponds to macular fixation. In fact, one may disregard the measurement of angle gamma and think of ocular deviation as the displacement of the corneal light reflex from the fixed corneal position assumed on monocular fixation. With both eyes open, binocular fixation would therefore imply the simultaneous registration of the corneal light reflexes on the monocularly fixating corneal points.

By thinking of epicanthus in terms of the corneal light reflexes, one notes the absence of true squint when the reflexes are found centered both monocularly and binocularly.

2. *False Macula and Amblyopia.*—False macula, as distinguished from abnormal retinal correspondence, is a form of amblyopia in which monocular foveal fixation is faulty. The corneal light reflex is thus seen to waver as the eye searches over a paramacular area to register imperfectly what the fovea is unable to accomplish. Fixation is unsteady, and the position of the corneal light reflex is not uniform. As a rule, it is displaced temporally in convergent squint and nasally in divergent squint. It may be confused with angle gamma when the light reflex is not centered, but the nature of such fixation should serve to distinguish these two conditions. In some cases the corneal light reflexes appear centered when both eyes are open, but alternate covering will show the nature of monocular fixation and reveal the existence of false macula. In other instances the light reflex may appear centered on attempted monocular fixation, whereas foveal fixation would yield a gamma corneal reflex.

In false macula with visible deviation in the amblyopic eye, one often finds that the eyes fail to respond to alternate covering and the poor eye remains deviated both with and without cover. In such cases testing the corneal light reflex (with prism) proves satisfactory for measuring the deviation. In convergent squint a base-out prism placed over the good eye will make that eye turn in in order to fix the light, and the poor eye will therefore turn out until the light reflex is centered on that cornea as well.

While one cannot judge visual acuity objectively by observing the corneal light reflex, one can with a little experience suspect amblyopia on the basis of the relative unsteadiness of the corneal light reflex in the amblyopic eye. This test is not infallible, but is occasionally helpful, especially when one is dealing with cases of malingering.

3. *Abnormal Retinal Correspondence.*—Abnormal retinal correspondence should not be confused with so-called false macula. In the former condition monocular fixation in either eye is not disturbed. However, a perverted relationship exists between the macula of one eye and the paramacula of the other whereby diplopia is avoided. This relationship is interchangeable and exists with either eye assuming the role of fixation.

Abnormal retinal correspondence is a frequent accompaniment of manifest squint. It differs from normal retinal correspondence in that bimacular fixation, or fusion, or superposition of images, does not occur when bimacular stimulation is made possible through artificial means, such as a prism, stereoscope or anglometer. One can detect abnormal retinal correspondence with any device which provides independent and simultaneous light stimulation to the two eyes

sufficiently strong to produce satisfactory corneal light reflexes. Measurement is based on movement of the lighted targets along a calibrated arc (as in the anglometer) or graduated bar (as in the stereosope) until they are in line with the respective fixing positions of the corneas. After the examiner notes the objective angle on the basis of such artificial centering of the corneal light reflexes and subsequent cessation of ocular movement on alternate flashing, the patient notes whether the images appear to touch. Approximation of these independent images at this deviation or objective angle speaks for normal retinal correspondence. Appreciable displacement of the images suggests an abnormal response, or so-called abnormal retinal correspondence, and the lighted targets are moved until their images approximate or cross at this new, or subjective, angle, and readings are again made. Thus in exotropia the images would appear to coincide not at the angle of the squint but at some angle nearer the "O," or primary position; like-

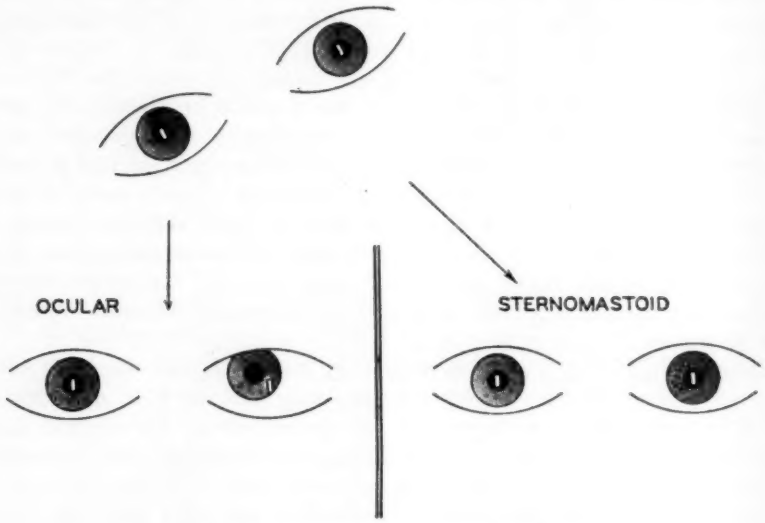

Fig. 7.—Ocular versus sternocleidomastoid torticollis.

wise, in esotropia the images appear to coincide at a less convergent angle than the deviation angle (fig. 7).

4. *Ocular Torticollis.*—Tilting of the head normally does not result in binocular imbalance or diplopia. In spite of such positional changes of the head, the corneal light reflexes remain centered. This objective manifestation of normal binocular responses proves helpful in indicating whether a head tilt is ocular or orthopedic in origin.

In a case of habitual head tilt, for the corneal light reflexes to remain centered when the head is straightened suggests that the condition is not ocular in origin. In ocular torticollis, the reflexes are centered only in the position in which the eyes can see binocularly. In other positions of the head, the light reflex is displaced in the nonfixing eye, and its position in relation to the position of the corneal reflex in the fixing eye suggests the nature of the diplopia and of the deviation.

5. *Cardinal Positions of the Eyes.*—Owing to the asymmetric conformation of the upper lids one is never certain from a cursory inspection of the eyes in the

oblique positions of gaze whether or not paralysis of a levator or a depressor muscle exists. Alternate covering of the eyes may not prove feasible, especially with an uncooperative patient. However, in observation of the corneal light reflex one is often surprised to learn at a glance that what appears to be a lag or paralysis of the superior rectus muscle is a false alarm due to the uneven conformation of the lids. This pseudosquint is analogous to the epicanthus simulating convergent squint.

In a routine examination with an ordinary flashlight, the movement of the examiner's eye when he is testing the eyes in the oblique positions proves clumsy, because his eye must keep moving behind the flashlight to obtain uniformity in response. However, in the Bielschowsky method, the head is tilted in different positions to produce the effects of oblique gaze, and the examiner can maintain his own head in a uniform position by operating the test light from a set position.

The cardinal anglometer lends itself quite satisfactorily to controlled measurement and inspection of the light reflexes for definite positions of gaze. Some patients may not be in a position to move their eyes obliquely upward or downward for more than a few degrees. It makes a difference if one's readings are based on an upward gaze of 30 or one of 10 degrees. With the anglometer one is in a position to note accurately the variations in imbalance with different selected positions of gaze, as well as the limits of oblique gaze. The head is kept fixed in a head rest, so as to prevent instinctive turning with movements of the eyes. Measurements can be made in but a fraction of the time required to make crude readings with prisms, and the test distance is fixed at 13 inches (33 cm.) in all directions of gaze. With prisms one is never certain of one's operating distance; instinctive head tilt cannot be properly controlled, and multiple prisms are not simple to manipulate. Also, with marked deviation the reliability of prism readings drops sharply, whereas angle readings conform to the angular deviations of the eyes along an arc.

6. *Fixing Eye.*—When a test light is brought gradually closer to the eyes so as to elicit convergence, a point is reached at which convergence is "broken" and one eye turns out and its reflex becomes displaced nasally. This is the nonfixing eye. When the corneal light reflex assumes a different position on alternate monocular fixation (with the cover test) than on binocular stimulation, one may assume that the eye which maintains the same position of the corneal light reflex with both binocular and monocular stimulation is the fixing eye.

7. *Near Point of Convergence.*—When a light is taken gradually nearer to the eyes an impulse to convergence is brought automatically into play, and one determines the proximal termination of the range at which the fixational corneal light reflexes can be maintained, or the near point of convergence. This is also the distance at which displacement of the reflex on one cornea occurs. The test is a simple objective one, and the examiner can note the "break" and foretell the nature of the diplopia.

8. *Prism Convergence and Prism Divergence* (fig. 8).—The amount of base-in or base-out prism which one can overcome with a rotary prism is a measure of the prism divergence or prism convergence. The prism reflex test lends itself satisfactorily to revealing to what extent the fixational corneal light reflexes can be maintained. A displacement of the light reflex from the fixation position as a result of an increase in the strength of the prism indicates a "break" in fusion and is of objective value in cases of suspected malingering when the fixational

reflexes are maintained in the presence of a moderate increase in the strength of the prism.

In routine prism tests one often relies on the patient to say when fusion is "broken" and, in overlooking the objective value of the prism reflex test, fails to realize that what appears to be fusion or compensation for the prism is in reality relative suppression in one eye. In such cases, the light reflex will keep shifting in the nonfixing eye as the strength of the prism is increased until stimulation of a more sensitive zone beyond the suppression area produces diplopia.

9. *Objective Heterophoria.*—A prism (say 10 prism diopters base down) placed vertically over one eye ordinarily causes vertical diplopia. The nature of such vertical diplopia depends on whether one is dealing with orthophoria, esophoria or exophoria. In orthophoria (fig. 9) the position of the eye is not changed by the

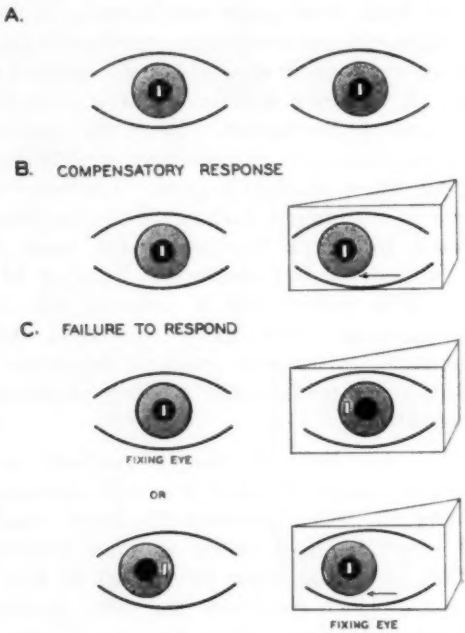

Fig. 8.—Prism convergence, with maintenance of fixational corneal light reflexes.

vertical prism, and the light reflex is displaced vertically, or in the direction of the apex. If the eye behind the prism is the nonfixing eye, the light reflex is higher and the false image created by the prism is also higher than the image seen by the fellow, or fixing, eye. If the patient transfers fixation to the higher image by turning the eyes upward, the light reflex becomes centered in the eye behind the prism and correspondingly lowered in the otherwise fixing eye. The displacement of the light reflex in either eye suggests diplopia, especially when alternate vertical fixation occurs. The nature of such displacement indicates the type of diplopia as well as the nature of the heterophoria.

In esophoria (fig. 10) the nonfixing eye turns in behind the vertical prism, and, in addition to vertical displacement of the corneal light reflex toward the apex by the prism, there is a temporal displacement as a result of the turning in of the eye. The position of the displaced light reflex indicates the nature of the

false projection and, incidentally, the type of heterophoria. From the standpoint of measurement, that amount of lateral prism which will bring the light reflex into a straight, vertical position or into an orthophoric position also serves as a measure of the amount of esophoria. By such vertical realinement of the dual reflexes the images likewise become vertically realined.

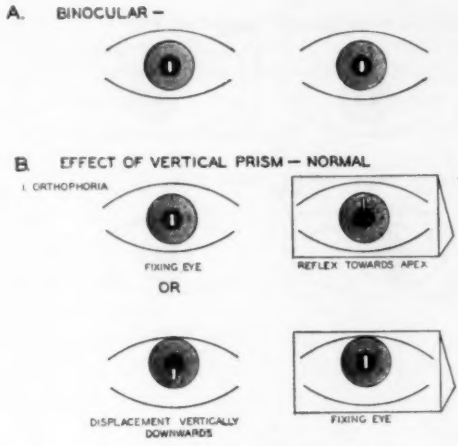

Fig. 9.—Vertical prism in orthophoria.

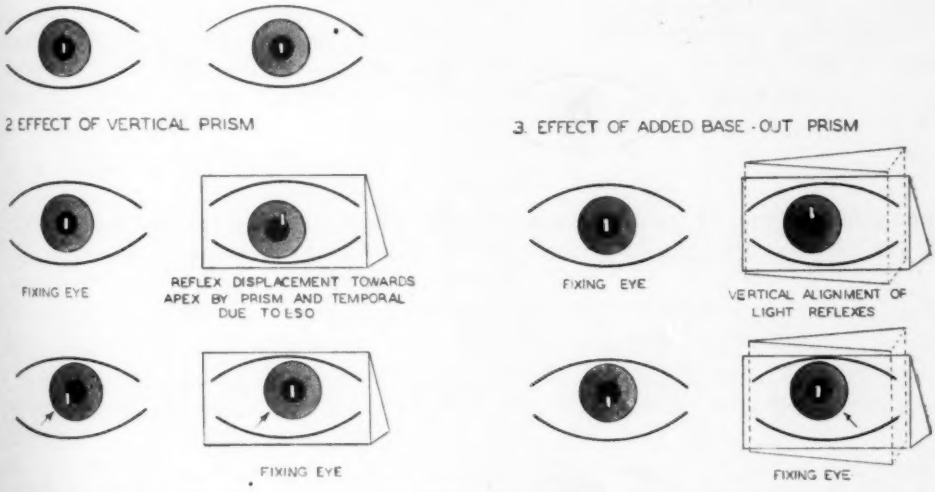
2 EFFECT OF VERTICAL PRISM
FIXING EYE
REFLEX DISPLACEMENT TOWARDS APEX BY PRISM AND TEMPORAL DUE TO ESO
FIXING EYE
3. EFFECT OF ADDED BASE-OUT PRISM
FIXING EYE
VERTICAL ALIGNMENT OF LIGHT REFLEXES
FIXING EYE

Fig. 10.—Esophoria and restoration of vertical corneal light reflexes by a neutralizing base-out prism.

In exophoria, the nonfixing eye turns out behind the prism, and the corneal light reflex is displaced inward as a result of the heterophoria and upward as a result of the vertical prism. In this condition, too, the amount of base-in prism required to restore vertical alinement of the dual light reflexes indicates the amount of exophoria. Also, the oblique positions of this displaced light reflex relative to the fixed position indicates the direction of projection of the false image.

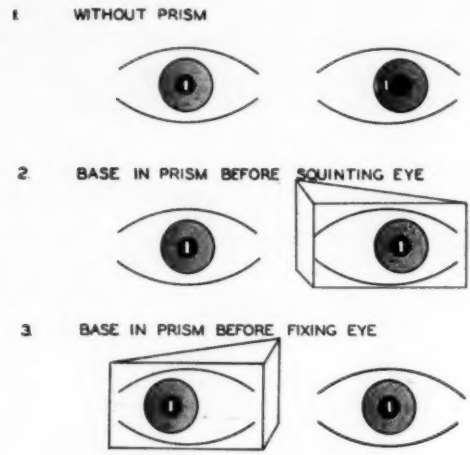

Fig. 11.—Divergent squint.

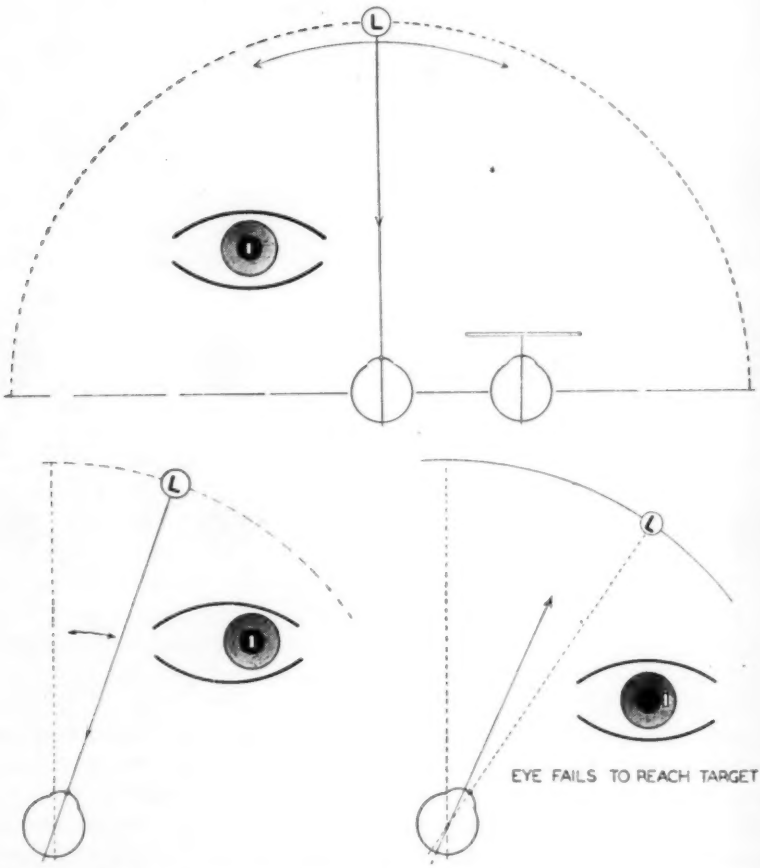

Fig. 12.—Range of monocular movement as shown by the corneal reflex test.

10. *To Distinguish a Heterophoria from a Heterotropia.*—In heterophoria, as distinguished from heterotropia, the eyes can respond to certain artificial stresses, such as those due to prisms, and still maintain binocular single vision and fixational corneal light reflexes. In heterophoria (fig. 11) the placement of a prism before the fixing eye will merely cause both eyes to turn in the direction of the apex of the prism (conjugate movement) in order for the fixing eye to maintain fixation behind the prism, and the position of the corneal light reflex will change only in the nonfixing eye; the placement of the prism before the nonfixing, or passive, eye will merely serve to move the light reflex in that eye, without altering the position or movement in either eye. In either condition, the amount of prism required to neutralize the deviation will also serve to bring the corneal light reflexes to the fixation positions. In heterophoria, each eye will overcome by the same amount the effects of a prism and still maintain binocular single vision (fixational corneal light reflexes).

11. *Paralytic Squint; Primary and Secondary Deviations.*—In paralytic squint one seeks to determine (*a*) the fixing eye, which has the fixational corneal light reflex; (*b*) the type and amount of displacement of the corneal light reflex in the fellow eye; (*c*) the changes in displacement of the light reflex in the nonfixing eye as the eyes are shifted in the cardinal directions of gaze, and (*d*) the amount of induced displacement in the otherwise fixing eye when the nonfixing eye is made to fix.

12. *Monocular Movements* (fig. 12).—Observation of the central corneal light reflex is invaluable in objective measurement of the amount of monocular motility.

With the anglometer, the patient's head is placed firmly in a head rest, and the testing eye is brought into a cyclopean position (the other eye covered). The lighted target is moved along the calibrated arc, and the observer notes through what range fixation of the corneal light reflex can be maintained, first in one direction and then in the other. The area of motility can be plotted so as to include both the vertical and the horizontal directions of gaze.

CONCLUSIONS

1. While the cover test rightfully serves as a basis for estimation of both latent and manifest deviations, it is not always easy for the examiner to observe slight movements in each eye on alternate covering.

2. Observation of the peripheral corneal light reflex (Hirschberg) is an unsatisfactory objective test, subject to the whims of the individual examiner.

3. Artificial restoration, or centering, of the corneal light reflexes to their fixation positions is a controlled and graded objective method for measuring ocular deviation and binocular imbalance.

4. Artificial centering of the corneal light reflexes can be obtained by acceptable methods which enable one to study binocular responses. Such methods include prism tests and use of the anglometer, phorometric stereoscope or synoptophore.

5. A prism refracts light toward the base, but displacement of the corneal light reflex takes place toward the apex. In squint, a correcting prism is so placed as to shift the corneal light reflex to the fixation position. The amount of prism required to center the corneal light reflexes also serves as a measure of the deviation.

6. The position of the peripheral corneal light reflex indicates the projection of the false image in the case of diplopia. To overcome diplopia, artificial centering of such reflexes is required. A prism placed with its apex in the direction of the deviation will restore the light reflexes to their fixation positions.

7. The prism reflex test simplifies the measurement of ocular deviation. It is intended to complement rather than to replace the familiar cover test, and is of objective value (*a*) in determining the amount of prism convergence and prism divergence as determined by the extent through which the fixational corneal light reflexes can be maintained with increasing prism, (*b*) in measuring heterophoria, (*c*) in distinguishing a heterophoria from a heterotropia, (*d*) in measuring squint with severe amblyopia and (*e*) in demonstrating malingering.

8. A rotary prism is the most satisfactory prism device for observation of the gradual correction from peripheral to fixational corneal light reflexes in manifest squint.

9. The refined Brewster stereoscope enables one to center the corneal light reflexes at the angle of deviation. Moreover, it can be adapted to different ranges of accommodation.

10. The anglometer provides a comprehensive picture of both the horizontal and the vertical deviation in any measured position of gaze through the incorporation of lighted carriers that can be adjusted to correspond to ocular deviations for these different positions.

11. The fixational corneal light reflexes are additionally helpful in the objective study of (*a*) the near point of convergence; (*b*) the fixing, or dominant, eye; (*c*) primary versus secondary deviation; (*d*) ocular torticollis versus torticollis originating in the sternocleidomastoid muscle; (*e*) the range of monocular movements; (*f*) binocular fixation; (*g*) abnormal retinal correspondence; (*h*) true squint versus pseudosquint (angle gamma), and (*i*) amblyopia.

QUANTITATIVE COMPARISON OF METHODS OF ADMINISTERING PHYSOSTIGMINE

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Physostigmine, because of its potent parasympathomimetic action, has retained an important position in ophthalmic therapy since first used as a miotic by Laqueur (1876).[1] It is administered usually in an aqueous solution and less frequently in an ointment or lamella. The official preparation is physostigmine salicylate, although the sulfate, which possesses similar pharmacologic properties, is also available.

This paper presents a comparison of the effectiveness of the various methods of local application, including iontophoresis and the use of a wetting agent.

In the selection of a method of administering a drug or in the adoption of a new method, its relative advantages and disadvantages must be carefully balanced. As part of the evaluation it is desirable to know the amount of drug entering the eye. Therefore a quantitative estimation was made of the physostigmine content of the aqueous humor of the rabbit's eye with different methods of application. No local or general deleterious effects were noted with any of the procedures described. A temporary corneal haze did, however, follow the iontophoretic experiments. It disappeared in one-half hour to one hour. No attempt was made to evaluate other factors, such as changes in vascularity, in tension and in the amount or content of the aqueous.

PRELIMINARY STUDIES

No accurate quantitative test for the chemical analysis of small amounts of physostigmine could be found. Biologic assay is the best method available. Pulewka [2] introduced the white mouse pupil as a test object for atropine bioassay and demonstrated its advantages. The superiority of the pupil of the white mouse over that of the cat was confirmed by Veit and Vogt [3] and by Orzechowski and Hundrieser.[4] Von Sallmann [5] used Pulewka's method for the estimation of atropine and scopolamine in different ocular structures. Salt solutions of these drugs were introduced iontophoretically by various routes. Investigation by Orzechowski and Hundrieser showed that the same method of bioassay was applicable to the quanti-

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2. Pulewka, P.: Das Auge der weissen Mausals pharmakologisches Testobjekt, Arch. f. exper. Path. u. Pharmakol. **168**:307, 1932; Ueber die Wertbestimmung von Heilmitteln, welche atropinartige Stoffe enthalten, ibid. **180**:119, 1935.

3. Veit, F., and Vogt, M.: Die Verteilung von Arneistoffen auf verschiedene Regionen des Zentralnervensystems, sugleich ein Beitrag zu ihrer quantitativen Mikrobestimmung im Gewebe, Arch. f. exper. Path u. Pharmakol. **178**:534, 1935.

4. Orzechowski, G., and Hundrieser, M.: Vergleichende Untersuchungen über Genalkaloide und entsprechende Alkaloide, Klin. Wchnschr. **15**:481, 1930.

5. von Sallmann, L.: Iontophoretic Introduction of Atropine and Scopolamine in the Rabbit Eye, Arch. Ophth. **29**:711 (May) 1943.

tative estimation of physostigmine. It utilizes a series of white mice whose pupillary reactions have been standardized by the subcutaneous or intraperitoneal injection of known quantities of drug. The amount of physostigmine present in solutions of unknown concentration can then be determined by a comparison of the pupillary responses to the standards.

White mice weighing 16 to 22 Gm. were used. The pupillary size was measured with a Leitz preparation microscope (ultrapaque) and ocular micrometer. A constant illumination was provided by a 6 volt tungsten bulb controlled by a rheostat and an ammeter in the electrical circuit. The minimum illumination that provided adequate visibility was chosen. The mouse pupil quickly assumed a stationary position that made possible an accurate measurement of the diameter.

The use of monochromatic light supplied by a red Wratten filter was tried in the hope of obtaining a light source that would not stimulate the rods of the mouse retina. A light source possessing this quality would give a larger pupil, increasing the range of contraction and therefore the accuracy of the test.[9] At the beginning of an exposure red light did not appear to induce miosis, but a slow contraction followed, introducing a difficulty not compensated by its advantages, and its use was discontinued.

Readings were taken between 11 a. m. and 3 p. m. to avoid the physiologic morning and evening dilation. The mice were kept in a quiet, dark room a minimum of one-half hour before each reading. A measurement of the normal pupillary size was made preliminary to each test. Uncooperative or excitable mice and those with a pupillary variation greater than 0.05 mm. from the average normal were not used. Animals were allowed a minimum of four days' rest between experiments.

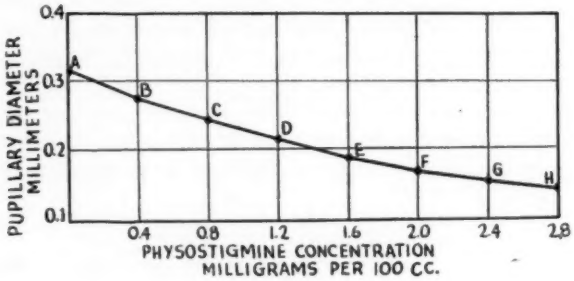

Chart 1.—Standard curve showing the pupillary diameter of rabbits after injection of various amounts of physostigmine salicylate in 0.25 cc. of distilled water. (*A* represents the pupillary diameter of a normal mouse which did not receive an injection, i. e. 0.31 mm.)

All solutions were used at room temperature. The injections were kept constant in quantity (0.25 cc.) and were introduced subcutaneously. After the injection of test solutions, a three-quarter hour interval, corresponding to the time of the drug's maximal action, was allowed before determinations were made.

STANDARDS

A standard curve was prepared representing the effect of injections of 0.25 cc. of distilled water containing, respectively, 0.001 to 0.007 mg. of physostigmine salicylate in gradients of 0.001 mg. All tests were repeated five times on groups of 5 mice each. The average normal pupillary diameter of each group measured 0.31 mm. Forty-five minutes later each pupil was again measured and the average for the 10 eyes determined.

The pupillary diameter produced by the injection of 0.001 mg. of physostigmine salicylate equaled 0.27 mm. Similarly, the diameter produced by 0.002 mg. equaled 0.24 mm.; by 0.003 mg., 0.21 mm.; by 0.004 mg., 0.19 mm.; by 0.005 mg., 0.17 mm.; by 0.006 mg., 0.15 mm., and by 0.007 mg., 0.14 mm. These results, plotted on a graph, are the basis for the standard curve illustrated in chart 1.

TECHNIC

Different methods of local administration of physostigmine were carried out on the eyes of albino rabbits. Each method was used five times. One hour after treatment 0.25 cc. of aqueous humor was removed, with the eye under pontocaine anesthesia, with a hypodermic syringe fitted with a 27 gage needle. This was injected subcutaneously into a mouse. For each method a group of 5 mice with an average normal pupillary diameter of 0.31 mm. was chosen. Forty-five minutes after injection the pupillary size of the mice was again measured and the average for the 10 eyes determined. The concentration of physostigmine was calculated by plotting the results against the standard curve, as shown in chart 2. In iontophoretic experiments a Birkhäuser tube electrode [6] was employed as the anode. The indifferent electrode was placed over the skull.

RESULTS

The administration of 0.1 cc. of a 0.25 per cent commercial physostigmine salicylate ointment produced a concentration in the aqueous humor of 0.3 mg. per hundred cubic centimeters.

One physostigmine salicylate ophthalmic lamella (0.11 mg.) gave a concentration of 0.74 mg. per hundred cubic centimeters.

One instillation of 2 drops (0.1 cc.) of 0.5 per cent physostigmine salicylate in distilled water brought about a concentration in the aqueous humor too small to be determined accurately. However, when this amount was given twice with a ten

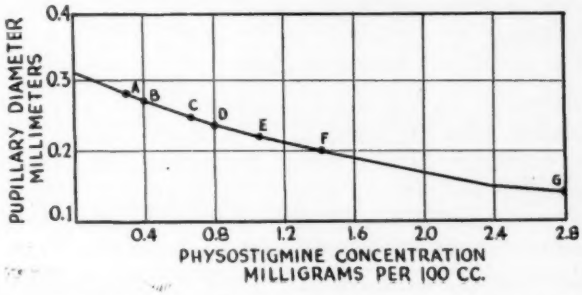

Chart 2.—The pupillary diameter after: *A*, 0.1 cc. of a 0.25 per cent physostigmine salicylate ointment was applied; *B*, 0.1 cc. of a 0.5 per cent solution of physostigmine salicylate in distilled water was instilled two times; *C*, one physostigmine salicylate lamella (0.11 mg.) was applied, 0.1 cc. of a 0.5 per cent solution of physostigmine salicylate in distilled water was instilled four times and iontophoresis with a 0.01 per cent solution of physostigmine salicylate in distilled water was carried out at 1 milliamperes for one minute; *D*, 0.1 cc. of a 0.5 per cent solution of physostigmine salicylate in a 1:5,000 solution of zephiran and 0.1 cc. of a 0.5 per cent solution of physostigmine sulfate in zephiran ophthalmic jelly were administered; *E*, 0.1 cc. of a 0.5 per cent solution of physostigmine sulfate in a 1:5,000 solution of zephiran was instilled four times; *F*, iontophoresis with a 0.01 per cent solution of physostigmine salicylate in distilled water was carried out at 2 milliamperes for two minutes and iontophoresis with a 0.1 per cent solution of physostigmine salicylate in distilled water was carried out at 1 milliampere for one minute, and *G*, iontophoresis with a 0.1 per cent solution of physostigmine salicylate in distilled water was carried out at 2 milliamperes for two minutes.

minute interval, the concentration was 0.4 mg. per hundred cubic centimeters. Four instillations at ten minute intervals resulted in a concentration of 0.68 mg.

The results obtained by O'Brien and Swan [7] with carbaminoylcholine chloride dissolved in a vehicle containing a surface tension-reducing agent stimulated a similar investigation with physostigmine. Physostigmine salicylate when dissolved

6. Birkhäuser, R.: Experimentelles und Klinisches zur iontophoretischen Behandlung von Hornhauttrübungen mit der Röhrenelektrode, Klin. Monatsbl. f. Augenh. **67**:536, 1921.

7. O'Brien, C. S., and Swan, K. C.: Carbaminoylcholine Chloride in the Treatment of Glaucoma Simplex, Arch. Ophth. **27**:253 (Feb.) 1942.

in distilled water containing the same wetting agent zephiran [8] in the same concentration, 1 : 5,000, yielded a cloudy precipitate. This wetting agent was found to be incompatible with salicylates. Substitution of the sulfate salt made a satisfactory mixture. This vehicle, when compared with distilled water alone, occasioned a significant increase in the concentration in the aqueous humor, a 2 drop instillation with a 0.5 per cent physostigmine sulfate solution producing a concentration of 0.8 mg. per hundred cubic centimeters and four similar instillations, a concentration of 1.08 mg. All instillations were followed by gentle massage of the cornea through the lids.

The administration of 0.1 cc. of a 0.5 per cent physostigmine sulfate solution in zephiran ophthalmic jelly produced a concentration of 0.76 mg. per hundred cubic centimeters.

Iontophoresis employing a 0.1 per cent of physostigmine salicylate in distilled water and a current of 1 milliampere for one minute resulted in a concentration of 1.46 mg. per hundred cubic centimeters. When the amount of current and the length of time were both doubled, the concentration increased to 2.8 mg. A 0.01 per cent solution gave correspondingly lower concentrations; i. e., 1 milliampere for one minute, a concentration of 0.62 mg., and 2 milliamperes for two minutes, 1.3 mg., per hundred cubic centimeters.

The addition of a wetting agent to the solutions used in iontophoresis did not increase their efficiency.

COMMENT

A general review of the literature advocating improved methods of ocular therapy in comparison with the standard form of instillation disclosed four main groups of agents: ointments or oily bases, ophthalmic disks or lamellas, solutions applied by iontophoresis and wetting agents. Quantitative bioassay of the aqueous humor of rabbits' eyes after administration of physostigmine with representatives of these four groups allowed the following deductions to be made:

In single treatments the ointment had a decided advantage over an aqueous solution. A lamella was twice as efficient as the ointment, and a surface tension-reducing solution or jelly corresponded in efficiency to the lamella. Iontophoresis was capable of producing concentrations three times as high as those produced by the lamella or the wetting agent.

The relative ease with which drops can be instilled warranted comparison of multiple instillations with iontophoresis. Application by means of an iontophoretic current of 1 milliampere for one minute of a 0.01 per cent solution of physostigmine salicylate was approximately equal in efficiency to four instillations (2 drops every ten minutes) of a 0.5 per cent aqueous solution or one instillation of 0.5 per cent physostigmine sulfate in a surface tension–reducing solution. Four instillations of a surface tension–reducing solution were approximately twice as effective as four instillations of a simple aqueous solution, but not so efficient as iontophoresis with a 2 milliampere current for two minutes and a 0.01 per cent solution or with a 1 milliampere current for one minute and a 0.1 per cent solution. When the current was increased to 2 milliamperes for two minutes, iontophoresis with a 0.1 per cent solution was approximately two and a half times as efficient as repeated instillations of 0.5 per cent physostigmine sulfate in a wetting agent.

In acute congestive glaucoma and in preoperative high intraocular tension, when it is important to lower the tension quickly, physostigmine is most valuable. However, its intensive use has been limited by unpleasant systemic symptoms.

8. Zephiran is a mixture of high-molecular alkyl, dimethyl and benzyl ammonium chlorides.

The addition of a wetting agent increased the efficiency of the physostigmine drops, but there was an accompanying increase of absorption into the general circulation through the conjunctiva and, unless the lacrimal apparatus was compressed, through the mucosa of the lacrimal sac and the nose as well. Careful application of only sufficient solution to cover the cornea or use of a corneal bath technic would minimize undesirable absorption.

Iontophoresis employing a corneal tube electrode presented a method of obtaining greater concentrations of physostigmine in the aqueous humor with a negligible amount of systemic reaction. Before it can be recommended for treatment, however, its action on normal and pathologic intraocular tension will require further investigation.

CONCLUSIONS

1. Bioassay was applicable to the estimation of physostigmine in aqueous humor.

2. One tenth cubic centimeter of a 0.25 per cent physostigmine salicylate ointment when applied to a rabbit's eye gave a higher concentration in the aqueous humor than an instillation of 0.1 cc. of a 0.5 per cent solution in distilled water.

3. A lamella containing 0.11 mg. of physostigmine salicylate was twice as efficient as the ointment.

4. There was no appreciable difference between 0.1 cc. of 0.5 per cent physostigmine sulfate as a vehicle for a water-soluble jelly and a solution when each contained a wetting agent. The concentration in the aqueous humor produced approximated that produced by the lamella.

5. Iontophoresis employing a 2 milliampere current for two minutes and a 0.1 per cent aqueous solution of physostigmine salicylate increased the concentration over that obtained by a 0.1 cc. instillation of 0.5 per cent physostigmine sulfate in a surface tension–reducing vehicle by three and a half times.

6. Four instillations of a 0.5 per cent physostigmine sulfate solution containing a wetting agent gave a concentration twice that obtained by four instillations of a 0.5 per cent aqueous solution but less than half that obtained by iontophoresis with a 0.1 per cent aqueous solution and a current of 2 milliamperes for two minutes.

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Ophthalmologic Reviews

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CLINICAL STUDY AND REVIEW OF TONOMETRY

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Several questions of interest arose in connection with tonometric measurements in the glaucoma clinic of the Long Island College Hospital (Dr. Henry Mitchell Smith, director). Would it be possible, for instance, to determine whether the reliability of the measurements would be enhanced by use of the Schiøtz instrument or by use of the McLean instrument? Could a table of equivalents be arranged with which to check one instrument against the other, so that, given a reading with one instrument, one could with reasonable certainty judge the probable reading with the other? And, finally, was one instrument more reliable than the other when used on eyes with low tension, normal eyes and eyes with high tension?

A study of 205 eyes was made, without full knowledge of the work of other observers, in an effort to solve these questions. It then seemed well to review the field of tonometry from Schiøtz' original contribution, in 1905,[1] which laid the groundwork and foundation for, and was actually the beginning of, modern tonometry. This review consisted mainly of an evaluation of the Schiøtz and McLean tonometers.

Much of this article will be elementary and some of it rather technical, particularly in connection with Friedenwald's plan of study and the work of Harrington and Parsons; most of it will be a review; some of the details may be of historical interest; perhaps the content will prove to be somewhat informative by merely refreshing the reader's knowledge of the subject.

It has been conceded, of course, that direct manometry is still the only scientifically accurate method of measuring the true intraocular pressure. However, this method being clinically inapplicable, it was necessary to devise an instrument for estimating the intraocular pressure by interpretation of measurements taken of the impressibility of the globe.

The development of the tonometer and the ingenuity exercised in its design are most interesting and have been ably reviewed by Lloyd.[2] From the beginning there has been a constant striving for greater accuracy in the interpretation of the instrument's measure of ocular impressibility.[3]

In 1868 Dor, in discussing one of the earliest models, gave expression to the following frequently repeated words, quoted by Lloyd[2]: "The tonometer in its present form is as yet so far removed from exactness that it cannot be regarded as mathematically correct, but on the contrary it is sufficiently exact for both practical and clinical use."

From the Department of Ophthalmology, Long Island College of Medicine.

Read at the staff conference of the Department of Ophthalmology, Long Island College Hospital, Oct. 28, 1942.

1. Schiøtz, H.: Ein neuer Tonometer, Arch. f. Augenh. **52**:401, 1905.
2. Lloyd, R. I.: Tonometry and the Various Instruments Employed in Its Practice, Am. J. Ophth. **13**:396 and 496, 1930.
3. Harrington, D. O., and Parsons, A. H.: Tonometric Standardization, Tr. Sect. Ophth., A. M. A., 1941, p. 203.

From that period to the present, approximately fifty different forms of tonometers have been devised, and within the last thirty years the form invented by Schiøtz has been the most generally used.[4] No doubt the ophthalmologist's requirements for clinical exactness have been greatly raised and he would no longer be satisfied with the crude model with which Dor worked. Nevertheless, as pointed out by Friedenwald comparatively recently, there has been little exact knowledge as to what, precisely, one is measuring when one uses the tonometer, nor has it been known what interpretation to give to the divergent readings obtained with the different types of instruments.

The first records of experiments in ophthalmomanometry were those of C. Weber in 1850, but he failed to draw any conclusions as to the normal intraocular tension. Wahlfors in 1888, using a manometer in connection with a normal living human eye, found the intraocular pressure to be 26 mm. of mercury. The usual method of determining the intraocular pressure experimentally has been to introduce a cannula into the interior of the eye and attach a manometer by means of tubing to the cannula. Both water and mercury have been employed in manometers, the choice depending on the construction of the apparatus. Among the early experimenters were Weber, Leber, Neisnamoff, Pfluger, von Hippel, Gruhagen, von Schulten, Stocker, Starling, Adamuk, Holtske, Hill and Flack. The experiments performed by these men were on dogs, rabbits and cats, and their conclusions were that the normal intraocular tension of these animals varies from 18 to 35 mm. of mercury. Weber and Wahlfors, according to McLean,[5] experimented on the human eye.

Before the days of the Schiøtz tonometer, estimations of pressure translated into millimeters of mercury were based on the experiment of Wahlfors. His determination of 26 mm. of mercury as the normal intraocular pressure was in harmony with the experiments on the lower animals made by other investigators. This was the only experiment on record in which an eye of normal tension had been connected directly to a manometer. When the Schiøtz tonometer was adopted, a new conception of the normal intraocular pressure was introduced.[5] With the advent of this instrument a point was reached at which the actual intraocular pressure of the average eye could be inferred with reasonable exactitude from a measure of its impressibility.[3]

The original model of the Schiøtz instrument is well known. The plunger is completely removable and has a concave surface at its point of corneal contact—continuous with the curve of the foot plate. Separate weights of 5.5, 7.5, 10 or 15 Gm. can be used as necessary, each being screwed, independently, on the center rod. The amount of dimpling of the cornea is transmitted to the lever and shown on the scale. By means of a graph or table the reading so obtained can be translated approximately into intraocular pressure in millimeters of mercury. Schiøtz found that the results were most reliable when the readings were made as near as possible to figure 3 on the scale. His original graph was based on readings obtained from the examination of 8 excised human eyes, each under eleven different manometric pressures; his findings at that time were that normal intraocular pressure varies from 15 to 25 mm. of mercury.

Schiøtz' experimental method, as described by Harrington and Parsons,[3] was briefly as follows: He connected the eye with a water manometer, and with a

4. Friedenwald, J. S.: Contribution to the Theory and Practice of Tonometry, Am. J. Ophth. **20**:985, 1937.

5. McLean, W.: Experimental Studies in Intra-Ocular Pressure and Tonometry, Arch. Ophth. **48**:23, 1919.

tonometer resting on the cornea measured the tonometer scale readings corresponding to different intraocular pressures. In each case two sets of readings were taken. First the connection between the eye and the manometer was left open. The pressure measured was the actual intraocular pressure with the tonometer in place. Second, the intraocular pressure was brought to a particular level, and the connection with the manometer was closed. The tonometer was then placed on the cornea and a reading made. Schiøtz used the results of the second method, which most closely approximates the clinical procedure in tonometry, to establish his nomogram, checking their accuracy by comparison with the results with the open manometer.

In 1915 Priestley Smith [6] stated the principle of the ocular tonometer as follows:

> The instrument measures the impressibility of the eyeball, and from the degree of impressibility we infer the intra-ocular pressure. The value of the inference is limited by the fact that the impressibility varies, not only with the intra-ocular pressure, but also with the size and structure of the eye.

He stated that in the early days of tonometry this limitation was clearly recognized, but that since the advent of the Schiøtz tonometer, which is more sensitive than any of its predecessors, it seemed to have been somewhat overlooked. He continued:

> It has become customary to translate the readings into millimeters of mercury according to the chart of averages, and to quote the figures as though they correctly represented the intra-ocular pressure in the individual case. Professor Schiötz, be it noted, has made no such claim for them. Even under the most favourable circumstances . . . the tonometer cannot tell us the pressure with precision. Eyes which have the same pressure will sometimes give different readings, and eyes which have different pressures will give the same readings. This we know. It is inherent in the nature of the case. What we do not know is the amount of error which may arise.

Priestley Smith's experiments were made chiefly on human eyes *in situ* in dead subjects and on excised eyes of pigs, the latter being obtained immediately after death. A few were made, for comparison, on excised eyes of sheep and bullocks.

He stated that if all human eyes were alike in size and shape, and if their tunics were equally pliable and extensible, it might be possible to construct a scale according to which an accurate tonometric reading would tell the intraocular pressure with precision in every case. But as eyes differ in these respects, a scale which is used in all cases must be wrong in some; even if strictly correct for the average eye, it will fail for individual eyes.

Two separate questions arose for consideration in connection with Priestley Smith's experiments: 1. With what degree of accuracy can one measure the impressibility of the eye? 2. With what degree of accuracy does the impressibility indicate the intraocular pressure?

As for the first, he stated that by applying a tonometer several times in succession to a given eye, taking care that the same internal pressure is reestablished (experimentally) before each application, one can learn the degree of accuracy with which the instrument does its work; if the readings differ materially, either the mechanism of the instrument or the manner of using it must be faulty. He hastened to add that the workmanship of the Schiøtz tonometer was admirable and that frequently in his first trials he got variations amounting to two and even three divisions on the scale owing to incorrect placing of the instrument.

6. Smith, P.: On the Limitations of the Tonometer, Ophth. Rev. **34**:65, 1915.

As for the second question—With what degree of accuracy does the impressibility indicate the intraocular pressure?—he stated that by applying the instrument to a number of different eyes in all of which the same internal pressure has been established, one can learn the extent to which the impressibility is influenced by conditions other than the height of the intraocular pressure. Accordingly, the amount of error likely to arise in a tonometric reading can be discovered by noting the extent to which the readings differ.

Priestley Smith concluded that

> . . . when a tonometric observation is recorded [with a Schiøtz instrument] it is the *reading* and not the *supposed equivalent* in mm. Hg that should be stated [for instance, 3/5.5 Gm. or 4/7.5 Gm.]. The reading is a fact; the other is an inference which may be correct or incorrect.
>
> Records in the form of mm. Hg are undoubtedly attractive, for they give an impression of scientific accuracy, but they are really a source of confusion and lead to false conceptions. . . .
>
> We are told that the pressure in healthy eyes varies from 13 to 27 mm. Hg. Whether this is true or not only the manometer could say for certain, but it is extremely improbable. It implies that not only the pressure of the intra-ocular fluid, but also that of the blood in the large veins of the retina and uveal tract, is twice as great in some eyes as in others, and all within healthy limits. Knowing as we do that the pressure in different eyes may seem to differ by 6 or 8 or even more mm. Hg, while in reality it differs not at all, we may well doubt the inference in question.

Priestley Smith stated that if his own observations were correct, the Schiøtz chart would place the intraocular pressure several millimeters of mercury too low. It should be kept in mind that these observations were recorded in 1915 and that Priestley Smith was referring to Schiøtz' original graph.

Schiøtz himself advocated and set the example of recording readings and not supposed equivalents, and Elschnig,[7] in thorough agreement, stated that pressure values in millimeters of mercury ought to be given only if the intraocular tension is exactly determined with a manometer.

Priestley Smith thoroughly supported the principle of the Schiøtz tonometer. He was careful to point out that his report was not an attack on this tonometer, but rather a defense of an admirable instrument against demands with which no instrument could possibly comply. He stated that the tonometer, though it cannot measure absolute pressure, can indicate changes of pressure with great precision. Applied to the same eye at different times, before and after an operation, before and after the use of a certain drug, at different hours of the day or night, and so forth, it can give invaluable aid in determining the occurrence of changes in pressure when these are doubtful. Again, as between the two eyes of the same person, when they are presumably alike in structure, it can be relied on to detect with certainty a difference of pressure much smaller than is discoverable by the finger.

When the eye of one person is compared with the eye of another or with a standard average, although considerable allowance must be made for the influence of structural differences, the tonometer is far more trustworthy than the finger as an index to the intraocular pressure.

In 1914 McLean [8] introduced his direct reading tonometer, and in 1919 [5] he gave an account of his work to place tonometry on a more accurate scientific basis. The report included certain observations regarding the normal intraocular pressure.

All that McLean had set out to do was to improve or eliminate several features in the Schiøtz and Gradle tonometers. The Gradle tonometer was devised in

7. Elschnig, A.: Designation of Tonometrically Ascertained Ocular Tension, Klin. Monatsbl. f. Augenh. **89**:289, 1932; Am. J. Ophth. **16**:8, 1933.

8. McLean, W.: Preliminary Report of a New Tonometer, J. Ophth., Otol. & Laryng. **20**:432, 1914.

1911.[9] It is essentially the Schiøtz instrument with several modifications. It is constructed more firmly; the handles are circular where grasped between the fingers; the corneal foot plate and stylet are smaller; the stylet is so attached that it cannot be dropped out of the tube in which it slides, and the four weights are each 1 Gm. being added to one another when the resistance of the hardened eyeball makes a heavier weight necessary. Every third marking on the recording scale is numbered in order to facilitate the tonometric reading. A chart is furnished which translates the tonometric readings into millimeters of mercury according to the empiric standard adopted by Schiøtz.

There were several respects in which McLean thought that the Schiøtz tonometer could be improved on. His criticisms were that (1) the reading scale of the instrument is too far away from the eye under examination, (2) the necessary translation of the reading into millimeters of mercury is cumbersome, (3) the chart supplied is a trying one to decipher, (4) the method of changing weights is difficult and time consuming and (5) the plunger fits the foot barrel so closely that capillary attraction may impair the accuracy of the reading.

The McLean instrument differs in many ways from those previously presented. The arc of the indicating needle is below instead of above, as in the Schiøtz and Gradle models, so that the numbered reading scale is as close as possible to the eye under examination. The scale is graduated in millimeters of mercurial pressure from 10 to 130, making possible a direct reading of the intraocular pressure, without recourse to graphs, and eliminating the need for noting millimeter or fractional millimeter oscillations. No weights are needed. The plunger is so supported that "no drag is produced by the capillary attraction of any fluid which may have remained on the cornea at the time of placing the instrument for a reading."

Several years were spent by McLean in experimentation to overcome mechanical difficulties before all was in readiness for the scale and the determination of the pressure according to the manometer. It was while attempting to check the accuracy of a testing apparatus with a Schiøtz tonometer that McLean discovered that the tonometer did not agree; neither did several other Schiøtz tonometers or a Gradle instrument. These tonometers gave lower readings than the manometer. It was then that experiments were conducted to investigate further the matter of normal intraocular pressure.

McLean's experimental results tended to revert to the standards established by Wahlfors and the early experimenters rather than the newer scale of the Schiøtz tonometer. They were performed on rabbits and dogs and on 3 living human eyes *in situ*. The 3 living eyes were all glaucomatous and were being removed on account of uncontrollable pain. The experiments on 2 were performed before McLean's tonometer was perfected. For the first eye, the measurements were 50 with the Schiøtz tonometer and 65 with the manometer. Tests made after enucleation disclosed the same variation. For the second eye, the measurement was 72 with the Schiøtz tonometer and 88 with the manometer. For the third, on which both tonometers were used, the Schiøtz instrument gave a measurement of 40, the McLean of 58 and the manometer of 60.

The present scale on the McLean tonometer was adopted on the basis of tests of nearly 200 enucleated eyes and a considerable number of eyes *in situ* of the lower animals. With this tonometer, the lower limit of normal intraocular pressure is to be considered as 22 and its upper limit not above 40. McLean stated that it is difficult to give an absolute limit of normal tension.

9. Gradle, H.: Modification of Schiøtz Tonometer, Ophth. Rec. **20**:19, 1911.

McLean [10] had joined Weber and Wahlfors and Wessely and Seidel as one of the only men who had done any experimental work on living human eyes by connecting the interior of the eye to a manometer.

During the course of the experiments on rabbits, an apparently new observation was made, namely, that the tonometer disclosed a marked difference in intraocular pressure with the eye under local and under general anesthesia. This was borne out by manometric readings, the tension dropping anywhere from 3 to 11 mm. during general anesthesia.

In all the experiments [5] on the living eye, both human and animal, the intraocular pressure was highest during local anesthesia, and as soon as the subject was thoroughly under the influence of ether, the pressure was lowered; the deeper the anesthesia, the lower the pressure, until a limit was reached. In animals it was noted that the pressure rose after the ether cone was removed, so that it was back to its original level as the animal regained consciousness. In the first of the cases of enucleation, the Schiøtz tension of the glaucomatous eye dropped from 75 under local anesthesia to 45 and that of the healthy eye from 19 to 10. In the second case, the tension of the glaucomatous eye dropped from 88 under local to 72 at the first reading under general anesthesia and to 47 as the anesthesia was increased.

Schiøtz' reply to McLean's criticisms of his instrument was as follows: 1. The distance between the reading scale and the eye matters but little. 2. Tonometric readings cannot be accurately translated into intraocular pressure in millimeters of mercury and it is preferable to state the weight used and the reading obtained, in the form of a fraction or otherwise, and to follow this with a figure designating the pressure deduced. McLean short-cut the calculations and arrived directly at the deduced pressure. 3. The chart can easily be enlarged. 4. It is a disadvantage to employ a constant weight, as in the McLean instrument; much more reliable indications are obtained by varying the weight according to the needs in the individual case. Schiøtz defended this thesis in considerable detail. 5. As to the influence of capillary action, he expressed the opinion that this factor is negligible; Elliot [11] agreed, stating that he had strong practical reasons for believing this opinion to be correct. Elliot's opinion, after using both instruments, was that the low position of the McLean scale is a distinct advance.

Dr. Marple, who claimed the distinction of bringing the first Schiøtz tonometer into this country, had become greatly interested and had been present at many of McLean's experiments. He requested the privilege of sending Dr. Schiøtz one of McLean's tonometers together with a diagram of the testing apparatus and the results of the tests he had seen. He desired Dr. Schiøtz' criticism of the apparatus and some explanation of why the results did not agree. In his reply, quoted by McLean,[10] Schiøtz failed to criticize the apparatus and the method of the testing, and his only comment relative to the experiments was, "Dr. McLean evidently has values different from mine. I do not understand why."

In 1920 Schiøtz [12] wrote that, although during the years since he had introduced his instrument he had never ceased to occupy himself with the tonometer, it had not been until the previous three or four years that he had been able to turn his full attention to a close investigation of the subject. His aim was partly to improve the instrument if possible, partly to extend existing knowledge of the mode of action

10. McLean, W.: Development of a Tonometer, Am. J. Ophth. **2**:417, 1919.
11. Elliot, R. H.: A Treatise on Glaucoma, ed. 2, New York, Oxford University Press, 1922.
12. Schiøtz, H.: Tonometry, Brit. J. Ophth. **4**:201 and 249, 1920.

of the tonometer and partly to check and correct the curves, the construction of which had been based on not more than 8 enucleated eyes.

Schiøtz conceded that, as might have been expected, the graphs based on measurements of 8 enucleated eyes were incorrect, and in 1924, utilizing the measurements of Priestley Smith on 12 dead eyes *in situ* and additional data he himself had obtained during the previous year on 30 dead eyes *in situ*, he compiled a new graph, which showed the readings in mercurial pressure to be 4 mm. higher than the computations on the graph accompanying the original instrument. Of the 30 eyes experimented on, 20 were in persons who had been dead for from eight to forty-eight hours prior to the measuring; on the remaining 10 the measurements were made one to two hours after death. Several measurements were taken for each pressure height. Schiøtz found no important difference between the two groups regarding the deflections, and he concluded that one may safely assume that for each degree of pressure the maximum and minimum deflections in living eyes will be about the same as in dead eyes. For these measurements Schiøtz used his old standard tonometer, which had been used for all his previous measurements and as the standard for the adjustment of other tonometers.

A deflection of 3 mm. (5.5 Gm. in weight) is the approximate limit for the normal intraocular pressure, and its mercury value is about 4 mm. higher than originally assumed. Schiøtz stressed the fact that to investigators who have recorded the deflections as the measure of the intraocular pressure, which is really the only correct method, the changes in mercury values are of no importance, because at present, as before, deflections of between 3 and 6 mm. or more signify the normal intraocular pressure. Those who have translated the deflection values into millimeters of mercury must rectify these according to the new graphs.

At the time that Schiøtz compiled his new graphs (in 1924),[13] he again emphasized the fact that a given deflection does not indicate a definite pressure in millimeters of mercury, that it represents a range between the maximum and minimum which is not insignificant. If, he stated, one fixes a deflection of 3 mm. (5.5 Gm. weight) as the limit between the normal and pathologic pressure, one does so because it represents a range between the normal and the pathologic. The boundary is never sharply defined. Consequently, with a deflection of 3 mm. (5.5 Gm.) the pressure will as a rule be about 29 mm. of mercury, but that deflection might be properly referable to the minimum graph and to an intraocular pressure of 26 mm. or to the maximum graph and a pressure of 33 mm. In other words, the deflection of the pointer does not indicate a precise pressure but a pressure *range*, which for the 5.5 Gm. weight is about 6 to 8 mm., for the 7.5 Gm. weight 8 to 10 mm., for the 10 Gm. weight about 13 mm. and for the 15 Gm. weight about 15 mm. Nor can it be otherwise, continued Schiøtz, when one takes into consideration the individual peculiarities in the size of the eyes and in the thickness and elasticity of their walls.

Priestley Smith's comment summarized the status of tonometry at the time: "We have here the limitation of the tonometer, and the fact cannot be altered as long as we employ the principle in question." Schiøtz[12] stated that he could not imagine any method available for living eyes by which the errors due to variations of the envelope could be eliminated.

In connection with this consideration of the variables in tonometry, Elliot[11] in 1922 wrote that there had been apparently unanimous recognition, by investigators who had carefully worked on the subject, of the profound influence, at least

13. Schiøtz, H.: Tonometry, Brit. J. Ophth. **9**:145, 1925.

theoretic, of the rigidity or elasticity of the tunic of the globe and of differences in the radius of curvature on the tonometric determinations. Where the authorities differ, he stated

> is in the matter of the extent to which this factor influences the readings obtained. Some observers think it to be of such great importance that it invalidates their scientific accuracy. McLean, on the other hand, whose admirable work on a large number of living eyes entitles his opinion to the greatest respect, does not agree with this assumption, and believes that, provided we have to do with a living eye, the overwhelming factor in the determination of the tonometer reading is the intra-ocular pressure, the condition of the tunic being comparatively unimportant. Obviously gross pathological change, such as calcification of the tunic, is excluded.

After working on the problem for a considerable time, Schiøtz constructed a new model of his instrument, presented in 1924,[13] in which the plunger and weight form one piece, which may be put into the cylinder from above. If a heavier weight is needed, another weight may be placed on top of the first one. This improvement eliminates the criticism often directed to the difficulty encountered in screwing on and off different weights for higher pressures. According to Schiøtz, the sliding cylinder with the small round disks has been replaced by one with ball bearings, making the cylinder more solid and the tonometer better balanced. In principle, however, the old model was retained.

During the course of his experiments, Schiøtz became aware of the extraordinary influence of the shape of the lower end of the plunger on the extent of the deflections of the pointer. This detail is elaborated on here because of several references that will be made subsequently. In the Schiøtz tonometer, the lower end of the plunger is concave, and the radius of curvature is the same as that of the concave surface of the base, viz., 15 mm. For technical reasons, the question was raised as to whether the lower end of the plunger should be made flat. Schiøtz recorded certain measurements obtained, respectively, with plungers having the ordinary concave end surface, a plane surface and a convex surface. They showed a surprising difference in the deflections of the pointer, especially as between the concave and the convex surface. This great difference, Schiøtz stated, was surprising but the explanation simple: In each case the plunger sinks into the membrane and depresses it until the depressed area is supported from below by a pressure that balances the weight of the plunger and its controlling parts; in each case the "supporting area" is the same but to obtain it the convex plunger must sink more deeply than the concave.

With a convex plunger and a relatively small weight one can deal with a large range of pressures, and Schiøtz stated in his earlier work that he ought perhaps to substitute a convex plunger for the concave in order to avoid the troublesome addition and removal of weights and the need for the corresponding four curves. On the other hand, he feared that the relatively small intervals between the deflections with the convex plunger in the case of high pressure would make the measurements less trustworthy and would result in objections to the proposed change.

However, Schiøtz was not inclined to abandon the idea of the convex plunger, for further experiments showed that just in the region with which one is most concerned the deflections are not smaller but considerably larger than with the concave. Additional studies were undertaken with the convex plunger in order to find the weight most suitable for clinical use; 5.2 Gm. for the plunger and its belongings was determined as the best. This weight gave a sufficiently large range, namely, from 22 mm. of mercury (deflection, 5.6 mm.) to 90 mm. (deflec-

tion, 0). And so, in 1927, Schiøtz introduced a tonometer with a convex plunger, which he named the Schiøtz x-tonometer.[14]

When comparative measurements with the two instruments were made on the same eye, Schiøtz found the readings equally constant with each, but when they were made on a number of different eyes it was noticeable that in cases in which the concave plunger gave uniform readings, the convex often showed slight variations which could hardly be attributed to error. He stated the belief that the small discrepancies were due to slight differences in the thickness and elasticity of the corneas, so small that the flatter surface of the concave plunger is not influenced by them, while the convex plunger, which sinks deeper and is more closely surrounded by the cornea, is sensitive to them. The flat plunger he believed to be undesirable because a slight error toward either convexity or concavity causes perceptible variations in the deflections.

After a thorough consideration of the question, Schiøtz concluded that for the time being at least it would be preferable to retain the plunger with the concave surface. He said [14]:

> This tonometer has now been tried for many years and has shown itself—*when correctly standardized*—to be a practical instrument for clinical use. The earlier disadvantage of having to screw the additional weights on and off is done away with, for the original weight of 5.5 grm. can now be added to by simply laying on it the 7.5 grm., 10 grm., or 15 grm. weight. . . .
>
> I must add, however, that the x-tonometer also is a good instrument, it gives a constant deflection and unquestionably it is safer for the corneal epithelium, especially in eyes with high pressure for which with the concave plunger one must use a proportionately high weight. Whatever shape be adopted, it is clear that the deflections given by different tonometers will not be comparable if the plungers are differently shaped.

The weight of the Schiøtz tonometer is represented by two distinct parts acting independently. The one consists of the cylinder, with its cup-shaped base, together with the loop carrying the flat strip and the graduated arc. This part of the tonometer weighs 10.1 Gm.[12] The other part consists of the plunger, with its loose weight, together with the pointer. The weight of this part varies with the loose weight employed. It is 5.5, 7.5, 10 or 15 Gm., respectively.

Schiøtz stated that the use of loose weights which have to be changed during the examination may appear inexpedient. Certainly it would be easier to use only one, but this would have to be considerably larger, an innovation which he considers absolutely wrong. A heavy weight means an unnecessary strain on normal eyes and causes a depression in the cornea of 1 mm. or more, which, Schiøtz wrote, is undesirable and according to his experience will not increase the reliability of the indications. His opinion is that the most reliable deflections are those from 1 to 5 mm. or, at most, 10 mm. If a deflection smaller than 1 mm. is obtained, a heavier weight should be employed, and if with a heavier weight the deflection is greater than 5 to 6 mm., a lighter weight should be substituted.

Schiøtz' declared that a heavy weight does not give exact information with regard to eyes with normal or slightly increased pressure; it causes fluid to be pressed out of the eye and thus reduces the pressure below its original state. He said that experimental measurements show that it is inadvisable to use weights that are heavier than is absolutely necessary for the production of reliable deflections of the pointer.

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14. Schiøtz, H.: Tonometry: A Tonometer with Convex Plunger, Brit. J. Ophth. **11**:116, 1927.

In this connection, it is interesting to note a report by Böck, Kronfeld and Stough,[15] that:

> The behavior of the intra-ocular tension of normal eyes in response to the application for two minutes of a [Schiötz] tonometer carrying a 15 Gm. weight was studied on fifty persons. In every case the tension dropped in proportion to the original tension. Normal eyes with tensions between 27 and 32 mm. showed a greater decrease than a small number of glaucomatous eyes with the same original tensions.

Friedenwald has stated that the actual expulsion of fluid from the ocular cavity cannot be instantaneous and may be reduced to a minimum by a reduction in the time of the tonometric reading to as short a period as possible.

In commenting on two tonometers made in America, Schiøtz [12] stated that the Gradle instrument is the Schiøtz tonometer except for a few "improvements" and that the McLean tonometer is

> a very beautiful instrument, but, to tell the truth, I cannot in this case either find any real difference between it and the Schiötz. The design and the principle are similar, though the index is made to point downwards instead of upwards. A few other slight alterations have been made, and these undoubtedly will influence the measurements, so that it will probably give results a little different from those obtained by the Schiötz tonometer. . . .
>
> McLean's tonometer subjects all eyes to the pressure of a considerable weight, viz: 25 grm., of which 12-15 grm. falls on the plunger. For most eyes this weight is unnecessarily heavy, more than 10 grm. being needed only in very rare cases. For reasons already given I consider it undesirable to graduate the scale in mm. Hg., as in the McLean tonometer.
>
> That Dr. McLean found the values obtained with the McLean and Schiötz tonometers to differ considerably is due, I think, in the first place to the fact that the Hg. values for the Schiötz were too low, and, secondly, in some degree, to differences in the relative weights of the various parts of the instruments and other differences. The tonometer is a very sensitive instrument, and the slightest alteration in any detail is likely to influence the deflections. For example, I have learnt recently that the lower end of the plunger in the McLean tonometer is convex.[16] I have already shown that the shape of the plunger has an important influence on the deflections. . . .
>
> The values [with the Schiötz and the McLean tonometer] can hardly be compared for they lie differently on the scale, but the instrument does not appear to offer any real improvement as regards reliability of measurement. The McLean tonometer is a Schiötz tonometer partly turned upside down, and as it is similar to the older instrument both in principle and build, it should measure as well, but nothing has been added which could lead us to expect it to measure better.

Schiøtz expressed his gratitude to Priestley Smith for his exposition of the Schiøtz tonometer and stated that he fully agreed with his conclusion that "It is not proved, and it is not likely, that the instrument errs to the extent that McLean's experiments suggest."

Elliot commented on Schiøtz' criticism of the McLean instrument on the ground of its high fixed weight and stated that McLean's experiments on living human eyes would seem to show that it is an accurate instrument when used on eyes with high pressure, whatever it may be on normal ones, and he added that clinical experience with the instrument serves to prove that its indications with regard to normal eyes can safely be relied on, for they are consistent with the other features present.

Elliot has used both the Schiøtz and the McLean tonometer, and he has expressed his confidence that either will prove invaluable to the surgeon who accustoms himself to its use.

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15. Böck, J.; Kronfeld, P. C., and Stough, J. T.: Effect on Intra-Ocular Tension of Corneal Massage with the Tonometer of Schiötz, Arch. Ophth. **11**:797 (May) 1934.

16. In a personal communication to the author, Dr. McLean wrote that the lower end of the plunger of his tonometer is flat.

TABLE 1.—*A Comparison of Pressure Readings Obtained with the Schiøtz and the McLean Tonometer (Millimeters of Mercury)*

Schiøtz Tonometer (Original Model)		McLean Tonometer	Schiøtz Tonometer (Original Model)		McLean Tonometer
Original Graph	1924 Graph		Original Graph	1924 Graph	
7	10	12	19	23	29
8	11	17	19	23	29
10	13	12	19	23	30
10	13	15	19	23	30
10	13	16	19	23	30
10	13	16	19	23	30
10	13	18	19	23	32
10	13	19			
10	13	20	20	24	28
			20	24	28
11	14.5	15	20	24	28
11	14.5	20	20	24	30
12	15.5	15			
12	15.5	15	20.5	24.5	29
12	15.5	16	20.5	24.5	29
12	15.5	16	20.5	24.5	29
12	15.5	18	20.5	24.5	30
12	15.5	18	20.5	24.5	30
12	15.5	18	20.5	24.5	30
12	15.5	18	20.5	24.5	31
12	15.5	18	20.5	24.5	31
12	15.5	19	20.5	24.5	32
12	15.5	19	20.5	24.5	32
12	15.5	19	20.5	24.5	33
12	15.5	20			
12	15.5	22	21	25	32
12	15.5	25			
			22	26	28
12.5	16.5	16	22	26	28
12.5	16.5	18	22	26	30
12.5	16.5	18	22	26	30
			22	26	30
13	17.5	18	22	26	31
13	17.5	19	22	26	31
13	17.5	19	22	26	31
13	17.5	20	22	26	31
13	17.5	22	22	26	31
13	17.5	23	22	26	32
13	17.5	23	22	26	32
13	17.5	25	22	26	33
			22	26	33
14.5	19	19	22	26	33
14.5	19	22	22	26	33.5
14.5	19	23	22	26	34
14.5	19	24	22	26	34
14.5	19	25	22	26	35
14.5	19	28	22	26	35
			22	26	35
16	20	21	22	26	35
16	20	22	22	26	35
16	20	23	22	26	36
16	20	26	22	26	36
16	20	26			
16	20	26	23	27	33
16	20	26	23	27	35
16	20	26			
16	20	26	24	28.5	32
16	20	26	24	28.5	32
16	20	27	24	28.5	32
16	20	27	24	28.5	35
16	20	27	24	28.5	35
16	20	28	24	28.5	35
16	20	28	24	28.5	36
16	20	28	24	28.5	37
			24	28.5	38
17	21	27	24	28.5	39
17	21	27			
			26	31	32
17.5	21.5	25	26	31	32
17.5	21.5	26	26	31	32
17.5	21.5	27	26	31	34
17.5	21.5	29	26	31	36
			26	31	36
18	22	24	26	31	36
18	22	27	26	31	36
18	22	28	26	31	36
			26	31	38
19	23	25	26	31	40
19	23	25	26	31	49
19	23	25	26	31	43
19	23	27			
19	23	27	28	32.5	40
19	23	27	28	32.5	40
19	23	27			
19	23	28.5	28.5	33	35
19	23	29	28.5	33	36
19	23	29	28.5	33	37
19	23	29	28.5	33	38

TABLE 1.—*A Comparison of Pressure Readings Obtained with the Schiøtz and the McLean Tonometer (Millimeters of Mercury)*—(Continued)

Schiøtz Tonometer (Original Model)		McLean Tonometer	Schiøtz Tonometer (Original Model)		McLean Tonometer
Original Graph	1924 Graph		Original Graph	1924 Graph	
28.5	33	39	40	45	53
28.5	33	39			
28.5	33	40	42	46	50
28.5	33	41	42	46	55
28.5	33	42	42	46	56
28.5	33	43	42	46	57
			42	46	60
29	33.5	38	42	46	62
29	33.5	40	42	46	63
31	35.5	34	43	47	55
31	35.5	40			
31	35.5	40	46	51	60
31	35.5	40	46	51	61
31	35.5	40	46	51	62
31	35.5	41			
31	35.5	42	50	54	65
31	35.5	43	50	54	70
33	37.5	48	56	60	65
33.5	38	45	60	64.5	98
33.5	38	50	60	64.5	105
34	39	39	65	69.5	90
			65	69.5	95
36	41	50			
			70	75	80
39	44	53	70	75	85
39	44	55			
39	44	55	89	..	110
39	44	55			

In summarizing his thoughts in regard to the choice of an instrument, James W. Smith [17] stated that every ophthalmologist who employs a standard tonometer has learned to know from experience what the normal reading is for his instrument and what constitutes an increase or decrease. He stated the belief that the controversy regarding the calibration of some instruments as compared with actual readings of intraocular pressure obtained with a manometer is chiefly of scientific and academic concern. It has been generally accepted that the relative readings of tonometry are of much greater practical importance than the absolute ones.

In 1932 Shope [18] undertook a study in tonometric measurements in a large series of cases in an effort to obviate the confusion resulting from the multiplicity of tonometers in general use and the interpretation of their readings. The purpose of this clinical investigation was to compile a table of comparative values for three popular tonometers, namely, the Schiøtz, the McLean and the Bailliart, giving a sufficient number of values to be practical, based on the application of the tonometer to human eyes.

The Bailliart tonometer was included in the study because, according to Shope, "This tonometer, because of its several new and practical features, has bid for a place in the ophthalmologist's armamentarium. These features include the direct reading in millimeters of mercury, no weights to change, and applicability in the erect posture and upon the sclera." Bailliart stated that his tonometer had been standardized with the Schiøtz instrument and adjusted to read the same, and it therefore seemed advisable to determine whether these two tonometers did read the same when applied to the same eyes.

Approximately 10,000 tonometric readings were taken for Shope's study. Of these, 1,687, taken on 220 eyes, were selected for further consideration. The others were rejected because the application of tonometers lowered the intraocular pressure, making parallel readings impossible.

17. Smith, J. W.: Tonometry, Am. J. Ophth. **9**:773, 1926.
18. Shope, P.: A Study in Tonometric Measurements, Am. J. Ophth. **15**:739, 1932.

A reading with a Schiøtz tonometer was obtained before and after each series of tension readings. Only those series were considered in which these two readings were the same or there was a reduction in tension not greater than one division on the Schiøtz scale.

The corneal curvature was determined in all cases with an ophthalmometer. No eyes were studied further which showed irregularities, an unusually high or low curvature or a large amount of astigmatism of the anterior surface of the cornea.

Although it was found that most eyes became softer after a tonometer was applied to the cornea a few times, many normal eyes, with a tension of 17 to 22 mm. of mercury (Schiøtz), did not become altered in intraocular tension by several applications of various tonometers. Many glaucomatous eyes became softer after a tonometer was applied a few times. The tension became less in practically all eyes after the McLean tonometer was applied two to four times. Priestley Smith has stated that the eye becomes softer after application of a tonometer by expression of fluid from the anterior chamber and that absence of this softening is a nearly constant indication of obstructive changes in the filtration angle. On the basis of his own observations, Shope concluded that apparently some factor other than the patency of the filtration angle must play a role in the softening.

In this study it was found that five Schiøtz tonometers, all in good repair, gave different readings when applied to the same eyes. Three of these were manufactured in Norway. Two were certified. The other two were of German manufacture. The readings with these tonometers occasionally differed as much as 15 mm. of mercury on an eye of normal intraocular tension and 15 to 20 mm. on a glaucomatous eye. Exact agreement was unusual.

With two Schiøtz tonometers and two McLean tonometers, a comparative study was made and tabulated. It was found that the equivalent values for these two kinds of tonometers were about as given by McLean and Schiøtz on the average, but for any given Schiøtz reading there was a considerable range in McLean readings.

Shope concluded that there is no exact parallel between readings obtained with the Schiøtz and with the McLean tonometer. For example, the McLean instrument gave a reading of 36 mm. for an eye for which the reading was 22 mm. with the Schiøtz tonometer; for another eye the values were 36 mm. with the McLean and 33 mm. with the Schiøtz instrument. For another eye the readings were 41 mm. with the McLean and 27.5 mm. with the Schiøtz, while still another eye registered 41 mm. with the McLean and 36 mm. with the Schiøtz tonometer.

Since a wide range of equivalents with one tonometer was found for any given reading with the other, no chart of exact or nearly exact equivalents could be drawn up, and only such a chart could have much practical value.

A series of tension readings was taken in which the Schiøtz and the Bailliart tonometer were compared. On the average, the Bailliart tonometer agreed fairly closely with the Schiøtz, the usual variation being 3 to 5 mm. of mercury. Occasionally large differences between the two were encountered; 1 eye registered 35 mm. with the Schiøtz and 16 mm. with the Bailliart.

Readings obtained with the Bailliart tonometer applied to the cornea with the patient erect and with the patient recumbent agreed or differed less than 3 mm. in 75 per cent of cases. Scleral readings with the Bailliart instrument were often surprisingly accurate, but just as often surprisingly inaccurate.

In the series reported on in the present paper, 410 measurements were taken on 205 eyes. One Schiøtz and one McLean tonometer were used. The Schiøtz

instrument was an original model of Norwegian manufacture. It has served me well for many years, and I have considered it reliable in its indications. Both instruments recorded properly when placed on their respective testing surfaces. In several instances the Schiøtz instrument was checked with a similar model of similar manufacture belonging to Dr. Henry Mitchell Smith, and on those occasions both instruments recorded alike or with negligible variation.

Corneal curvature was not measured, but no eyes were included that showed extensive corneal scarring. A tonometric measurement was taken with the Schiøtz instrument first, and this was followed by a reading with the McLean tonometer after one minute. All readings were taken by me.

As will be noted in table 1, for practically every reading with the Schiøtz instrument there was a range of readings with the McLean. For instance, 16 eyes that measured 20 mm. of mercury Schiøtz (1924 graph) gave readings of from 21 to 28 mm. McLean. Twenty-five eyes measured 26 mm. Schiøtz and 28 to 36 mm.

TABLE 2.—*Summary of Pressure Readings Obtained with the Schiøtz and the McLean Tonometer (Millimeters of Mercury)*

Number of Eyes	Schiøtz Tonometer		McLean Tonometer	Number of Eyes	Schiøtz Tonometer		McLean Tonometer
	Original Graph	1924 Graph			Original Graph	1924 Graph	
1	7	10	12	10	28.5	33	35-43
1	8	11	17	2	29	33.5	38-40
7	10	13	12-20	8	31	35.5	34-43
3	11	14.5	15-20	1	33	37.5	48
15	12	15.5	15-25	2	33.5	38	45-50
3	12.5	16.5	16-18	1	34	38	39
8	13	17.5	18-25	1	36	41	50
6	14.5	19	19-28	4	39	44	53-55
16	16	20	21-28	1	40	45	53
2	17	21	27	7	42	46	50-63
4	17.5	21.5	25-29	1	43	47	55
3	18	22	24-28	3	46	51	60-62
18	19	23	25-32	2	50	54	65-70
4	20	24	23-30	1	56	60	65
11	20.5	24.5	29-33	2	60	64.5	98-105
1	21	25	32	2	65	69.5	90-95
25	22	26	28-36	2	70	75	80-85
2	23	27	33-35	1	89	..	110
10	24	28.5	32-39				
13	26	31	32-43	205			
2	28	32.5	40				

McLean. Seven eyes that measured 46 mm. Schiøtz gave readings of from 50 to 63 mm. McLean. There were similar ranges of values in practically all the other groups. Conversely, for practically every reading with the McLean instrument, there was a range of readings with the Schiøtz.

The readings on this series of eyes tend to show that the equivalent values of the two tonometers are about as given by McLean and Schiøtz. For instance, 18 eyes that measured 23 mm. of mercury Schiøtz (1924 graph) showed an equivalent of 25 to 32 mm. McLean (table 2). This is well within the average normal for both instruments. Twenty-five eyes that measured 26 mm. Schiøtz showed an equivalent of 28 to 36 mm. McLean. This is still within the normal range. Ten eyes measured 28.5 mm. Schiøtz, with an equivalent of 32 to 39 mm. McLean. At this point one begins to reach the approximate upper limits of normal for both instruments. Thirteen eyes measured 31 mm. Schiøtz and 32 to 43 mm. McLean, and 10 eyes measured 33 mm. Schiøtz and 35 to 43 mm. McLean. The last 2 Schiøtz readings, 31 and 33, are somewhat above the upper limit of normal, 29 mm., and, similarly, the McLean readings have passed the recognized upper

limit of normal, 40 mm. As one gets to the higher Schiøtz readings, the McLean equivalents are also definitely above normal. One eye measured 39 mm. with both instruments.

On the basis of this data, one can only subscribe to the conclusions reached by Shope in his study.

Shope called attention to the tenet already recognized almost unanimously as fundamental, that no tonometer may be expected to give a definite idea of intraocular pressure until the variables which enter into tonometry can be controlled or calculated. He listed the principal variables as follows: (1) impressibility of the cornea, (2) distensibility of the corneoscleral coat, (3) compressibility of the choroid, (4) freedom of outflow of the aqueous, (5) amount and compressibility of the orbital fat, (6) corneal curvature and (7) size of the globe, as, other things being equal, a large globe will distend more than a small one.

Harrington and Parsons have more recently regrouped into the following classification the factors which, aside from the actual intraocular pressure, determine a tonometric reading: (1) rigidity of the ocular coats, (2) volume of corneal indentation produced by the tonometer plunger, (3) distortion of the cornea by the tonometer foot plate irrespective of indentation by the plunger, (4) expulsion of intraocular fluid by the weight and by the manner of application of the tonometer and (5) mechanical accuracy of the tonometer as to (*a*) adherence to specifications and (*b*) reduction of reading errors.

Shope also called attention to several possible causes of error that may decidedly influence the accuracy of a tonometric reading. It might be well to keep them in mind: (1) tonometer not applied to the corneal center, (2) tonometer not erect, (3) fingers pressing on the globe, (4) lids touching the cup-shaped base, (5) patient squeezing the eyelids, (6) unsteadiness of fixation of the eye, (7) lack of correction for movements of the eye when it is covered, the movements being due to heterophoria, and (8) pressing down or lifting up on the handle of the Schiøtz or McLean tonometer (this is regulated on the Bailliart instrument).

In 1936 Adler, Berner and Meyer [19] reported the results of a comparison of tonometers for their clinical accuracy. They devised an instrument for the testing and compared the readings of different tonometers with actual intraocular pressures and the readings of standard tonometers of the same make at different pressures. Schiøtz, McLean and Gradle tonometers were found to be sufficiently accurate for clinical purposes. Between the pressures of 20 and 40 mm. of mercury there was rarely a difference of more than 4 mm. between the actual pressure and the tonometric reading. One Bailliart tonometer was tested and found entirely inaccurate. McLean readings between 20 and 30 mm. were 3 mm. higher than the Schiøtz and between 30 and 40 mm. were 4 mm. higher than the Schiøtz.

Four Schiøtz instruments were taken at random and compared. For pressures between 20 and 30 mm., there was no more than 3 mm. difference in the readings. At higher pressures, greater inaccuracies developed, but the inaccuracy was constant for the particular instrument and when known could be allowed for in the readings.

The authors stated that for accurate readings the plunger of the tonometer must be absolutely clean, the foot plate must rest smoothly on the cornea approximately on the center and the tilt of the instrument must not be more than 15 degrees. Weights for the Schiøtz instrument should be selected so that the pointer reads between 3 and 7.

19. Adler, F. H.; Berner, G. E., and Meyer, G. P.: A Comparison of Tonometers for Their Clinical Accuracy: Summary (A. G. Fewell), Am. J. Ophth. **19**:49, 1936.

Friedenwald [4] in 1937, in a brilliant "Contribution to the Theory and Practice of Tonometry," presented an analysis of his studies of the structural rigidity of the eyeball and the influence of this variable in tonometry. This comprehensive and for the most part mathematical treatise was unquestionably the most important advance in many years in the field in which there has been a "relative paucity of original studies and of new developments." [20] As a result of this study, as pointed out by Harrington and Parsons, three of the major variables in a tonometric reading have so far as theoretically possible been mathematically reduced to constants. The potential accuracy of impression tonometry has thus been greatly increased.

Friedenwald reemphasized that tonometric measurements are not direct measurements of intraocular pressure but are measurements of the degree of indentation of the cornea by the tonometric plunger with a given load. As was noted by the earliest writers on tonometry, this measurement is not dependent on the intraocular pressure alone but is influenced, as has already been stated, by other factors, of which the distensibility of the eyeball as a whole is the most important.

Friedenwald's purpose was to obtain if possible some measure of this resistance to deformation and some indication of the appropriate corrections in pressure readings which variations in such resistance should dictate. "If," he stated, "we compare two eyes which actually have the same intraocular pressure but which differ markedly in the rigidity or distensibility of their coats, we will erroneously conclude from the tonometric reading that the pressure in the more rigid eye is higher than in the more distensible eye." This study represented an attempt to disentangle these two factors in the tonometric reading.[21]

For it Friedenwald used the Schiøtz tonometer with its original concave plunger end of the same radius of curvature as the tonometer foot plate; he omitted all consideration of the rounded plunger of the Schiøtz x-tonometer and of the plunger of the McLean [16] instrument. He stated, first, that more extensive experimental and clinical data exist for this type of plunger and, second, that the theoretic development of the relations which he sought are simpler when this form is considered.

When the Schiøtz tonometer is placed on the eye, a twofold effect is produced. Since the foot plate has a radius of curvature of 15 mm., while the radius of curvature of the cornea in normal eyes varies from 7 to 8 mm., the cornea must be flattened over the area of contact with the tonometer. In addition, the plunger indents the cornea, and it is the depth of the indentation from the level of the foot plate that is read on the tonometer scale.

"When the plunger indents the cornea," the author continued, "fluid is displaced from beneath it, and the intraocular pressure is raised. Part of the displaced fluid may be accounted for by an expulsion of blood from the intraocular vascular bed and an actual absorption of fluid out of the ocular cavity, while the remainder can be accommodated only by a distortion of the eyeball as a whole, no doubt mainly by an expansion in the equatorial zone of the eye."

Friedenwald stated that experimental work has shown that a measure of the compressibility of the intraocular vascular bed is included in the elasticity measurement, while the expulsion of intraocular fluid from the interior of the eye, as a result of the massaging effect of the tonometer, does not introduce important errors if the tonometric measurements are done rapidly.

20. Terry, T. L., in discussion on Harrington and Parson,[3] p. 228.

21. Friedenwald, J. S.: Contribution to the Theory and Practice of Tonometry: Condensed Summary (A. G. Fewell), Am. J. Ophth. **21**:193, 1938.

Perhaps it would be best to quote Friedenwald directly in regard to the general plan of his study:

> We have first attempted a theoretical analysis of the factors involved in a tonometric reading and have grouped together for consideration those factors other than pressure which may be thought of as contributing to the resistance that the eyeball exhibits to indentation by the tonometer. Previous studies on the elasticiy of the eye are then considered, and from the experimental data recorded by other observers a mathematical law is derived of the relation between volume and pressure changes in the eye, together with a mathematical formulation of what may be regarded as the coefficient of rigidity of an eyeball independent of pressure. This law is then applied to tonometric readings on the assumption that the application of the tonometer with successively different weights to an eye produces in the eye successively different pressure and volume changes. In order to accomplish this, it is necessary to know the volume of the corneal indentation produced by the tonometer plunger, and the intraocular pressure at the time when the tonometer is resting on the eye. The latter can be computed directly from the data given by Schiötz in the original measurements which he made in order to calibrate his tonometer. The former may be calculated to a fair approximation. . . . It follows that with the aid of these calculations the coefficient of rigidity of an eye can be computed from a pair of tonometric measurements made with two different weights. By allowing for the distortion of the cornea produced by the tonometer foot plate, the intraocular pressure that existed before the application of the tonometer on the eye may be estimated. A chart or nomogram has been constructed with the aid of which these otherwise laborious calculations can be performed graphically.
>
> The second stage of the investigation has consisted in applying these methods for the measurement of the rigidity and pressure first to normal and then to abnormal eyes. A statistical analysis of the results on normal eyes has enabled us to evaluate the effect of age, sex, axial refraction, and corneal curvature on intraocular pressure and ocular rigidity as determined by this method. The average normal ocular rigidity is found to be somewhat lower than that implied in the data used by Schiötz to calibrate his tonometer, and the average normal intraocular pressure is found to be correspondingly higher than that assumed by Schiötz. Using these data, a correction for the Schiötz chart or nomogram is obtained. Finally, the results of measurements of ocular rigidity and pressure in certain diseases are presented, and also an analysis of the effect of certain drugs on the rigidity and pressure of the eye.

Friedenwald based his experimental work on the data reported by Schulter, Koster, Schiøtz, Ridley and Clark on the distensibility of the eyeball and on the experimental data which Schiøtz used in computing his 1924 scale of pressures. In regard to the latter, Friedenwald stated, "The extraordinary perspicacity with which Schiötz analyzed his own results has been a recurring source of help to us in our study." Harrington and Parsons, also commenting on the experimental data supplied by Schiøtz, stated that the final measurements and calculations from which Schiøtz computed his 1924 scale of pressures are models of scientific care.

By using the data on which Schiøtz based his tonometric chart, Friedenwald computed the pressure which exists within the eye at each tonometric scale reading for each weight of the tonometer plunger and also the volume of the indentation corresponding to each tonometric scale reading. With the aid of these calculations and an empiric formula for the relation between pressure and volume changes in a given eye based on the experimental work on ocular distensibility, a nomogram was developed from which the coefficient of an eye may be computed from two tonometric readings with two different weights. (Measurements were made routinely on normal eyes with the 5.5 and 10 Gm. weights and in the cases of elevated tension with the 7.5 and 15 Gm. weights. In a few instances all four weights were used.) Finally, by allowing for the distortion of the cornea due to the tonometer foot plate irrespective of the indentation produced by the plunger, a method was developed for calculating the true intraocular pressure free from errors due to the rigidity of the ocular coats.

Having determined the average rigidity coefficient of normal emmetropic eyes, Friedenwald was in a position to recompute the Schiøtz scale so that tension read-

ings made with its aid would be accurate, at least when the ocular rigidity was normal. This chart is based on the assumption that the tonometer used is, in fact, an accurate mechanical replica of the standard Schiøtz tonometer.

The average normal pressure determined with such an instrument by means of this chart will be found to be 25 mm. of mercury. Readings above 35 mm. are to be attributed to either abnormally high pressure or abnormally high rigidity. If measurements on an eye are made with two different weights and the same pressure is indicated on the chart with each weight, the eye may be considered as having normal rigidity and the pressure reading as accurate. If with the heavier of the two weights the apparent pressure reading is higher, the eye is abnormally rigid, and the actual intraocular pressure is lower than either of the two readings. If with the heavier weight the apparent pressure reading is lower, then the eye is abnormally distensible, and the actual intraocular pressure is higher than either of the two readings would indicate.

Friedenwald found that the rigidity of the eye is increased in old age and in extreme myopia. This is likewise the case in intraocular inflammatory disease and not infrequently gives rise to an erroneous diagnosis of secondary glaucoma. In primary glaucoma, the ocular rigidity tends to be increased by long-continued high pressure. The effect of certain drugs on the ocular rigidity has been studied, and it was found that those drugs which act as vasodilators tend to diminish ocular rigidity and those which act as vasoconstrictors tend to increase ocular rigidity. The cause of the relative increase in the rigidity of the eyeball in extreme myopia has not been ascertained. The possibility that the sclera has been stretched beyond its elastic limit is suggested.

Harrington and Parsons [3] supplemented the work of Friedenwald and in 1941 presented a paper on a method of increasing the accuracy of tonometry. This valuable study will undoubtedly go far toward achieving the long sought goal of tonometric standardization.

Following in the footsteps of Schiøtz and Friedenwald, who, they declared, have done most to point out the need for tonometric standardization, Harrington and Parsons stated:

> We have made a searching analysis of Friedenwald's work and are entirely in agreement with his theory and his conclusions, all of which we have accepted in toto as logical and in definite order. We feel, however, that the work exhibits several drawbacks which have robbed it of some of the appreciation that should have been its due, and while we are unable to add to the fundamental concepts involved it seems highly desirable to make better use of these concepts by presenting them in somewhat less abstruse form and in such a way as to make them easily applicable to clinical tonometry.
>
> We make no attempt within the limits of this paper to review the vast subject of tonometry. . . . We are content with the more modest aim of using the salient points presented by various authors to evolve a clinically workable method of tonometry and one which we feel is more accurate in its interpretation of tonometric readings. . . . Schiötz' experimental work is readily available, but it remained for Friedenwald to coordinate it in such a manner as to eliminate the important variable of the resistance to pressure which is exerted by the rigidity of the ocular envelope and thus make it possible to determine to a reasonable degree of approximation the actual resistance to tonometric pressure exerted by the intraocular pressure alone. The assumption of an average rigidity of ocular coats thus becomes inadmissible. . . .
>
> Friedenwald's work has served to emphasize the fundamental accuracy of the findings of Schiötz. If one accepts the validity of the theory that ocular rigidity is a prime factor in the determination of actual intraocular pressure, one must automatically accept the principle of the tonometer with multiple weights and a concave plunger. Schiötz anticipated us in this when he convinced himself that his own X tonometer added nothing of value sufficient to warrant its adoption.
>
> Clinical application of the increased accuracy of interpretation contributed by Friedenwald to the Schiötz method, plus the vast accumulation of clinical data based on the Schiötz scale,

plus the practically international acceptance of the Schiötz nomogram, provides a widely used, clinically accurate method of impression tonometry as capable of rigid standardization and universal adoption as the Snellen visual acuity test. It remains but to devise an instrument by which this method may be fully utilized.

As has already been noted, Schiøtz was much distressed by the tendency to record tonometric readings in terms of millimeters of mercury. Harrington and Parsons said:

In spite of his exhortations the clinical use of direct pressure recording has persisted and, it cannot be denied, has much to be said in its favor. This attempt at a more direct approach to the analysis of intraocular pressure has, through wide clinical use and common consent, succeeded gradually in almost replacing the original Schiötz method. It is largely responsible for the search for satisfactory direct reading instruments, such as those designed by McLean and Bailliart.

It is indirectly responsible for the confusion and lack of standardization in tonometry today.

The authors stated that the Schiøtz tonometer in its present form is not a standardized instrument in any sense of the word. They carefully checked the weights and measurements of a large number of such tonometers, some manufactured in Europe and some in the United States. They found no instrument that adhered to Schiøtz' original specifications and no two instruments that were alike in more than the diameter of the plunger.

Harrington and Parsons undertook to analyze, simplify and recalculate what they termed the fundamentally sound principles presented by Friedenwald, in order to make them practical for clinical application. As a substitute for the graph method of calculating ocular pressure and rigidity, as proposed by Friedenwald, they presented a simple numerical chart from which the true intraocular pressure may be read when two tonometric readings are taken, each with a separate weight. In addition, they presented a new tonometer, designed to minimize reading errors, adhering rigidly to fundamental specifications and capable of use as a multiple weight instrument according to the directions of Schiøtz or, with the new numerical chart, according to the method of Friedenwald. It is also, within the natural limits of any multiple weight tonometer, a direct reading instrument.

Friedenwald [22] agreed that the numerical chart has much to be said for it in respect to convenience, but he cautioned that such a table of numbers may readily give a false sense of exactitude. One must keep constantly in mind, he counseled, the range of probable error in the measurement. As for the new instrument, he stated:

I have had the opportunity to see a photograph of Dr. Harrington's tonometer and have been much impressed with its appearance. I have not had an opportunity to study the instrument itself and should prefer to reserve judgment about it. There can be no doubt, however, that the authors are decidedly on the right track. The slovenliness in construction of Schiötz type tonometers sold at a substantial price in this country is really shocking. I once weighed half a dozen instruments all produced by the same manufacturer and found variations of 2 Gm. or more in the weight of the parts which rest on the tonometer foot plate and of over 1 Gm. in the weight of the plunger and the lever arm. It would seem that the manufacturer is unaware that the mark 5.5 which he has engraved on the instrument means 5.5 Gm. . . .

I believe that the authors will agree with me in the assertion that standardization of the tonometer should include rigorous definitions of the permissible variations from the standard. . . .

Further desiderata are as follows:

The scale should be close to the eye, as in the McLean tonometer. The center of gravity of the whole instrument should be low. The plunger should have the concave end and variable weight of the Schiötz model rather than the convex end of the Schiötz X model and of the

22. Friedenwald, J. S., in discussion on Harrington and Parsons,[3] p. 224.

McLean[16] instrument. In addition, the instrument should be rugged and easily disassembled for cleansing, with its more delicate moving parts enclosed and protected. It should be reasonably inexpensive.

It is evident that many of these requirements are fulfilled by the instrument shown.

T. L. Terry,[20] in discussing the paper of Harrington and Parsons stated that it would not be difficult to raise objections and criticisms, chiefly because impression tonometry, or for that matter any form of tonometry, is through necessity not scientifically accurate, especially if the tension reading is taken directly in terms of intraocular pressure. Perhaps the foot plate is not curved in the best form; perhaps the plunger should be larger or smaller for best results.

He stated that the perfect solution of the problem of determining the actual intraocular pressure as a clinical test lies in the development of the best substitute for manometric determination, a solution which would require in combination a physiologist, a physicist and an expert mechanic skilled in the minute accuracies of the watch maker. He said:

> True, the Schiötz type of instrument is the most popular and does seem to be the most satisfactory; yet if as much time and effort were spent on study and use of the other general types, as represented by the Souter, the aplanation and the recoil tonometer, perhaps they would be found as good or better. . . . Harrington and Parsons are to be complimented for designing a new impression tonometer and for explaining its apparent superiority with broad consideration of the many obstacles incident to tonometry. Such work as theirs requires the utmost patience, time and skill. . . . The instrument . . . will be its own best critic once it has been tested thoroughly by critical clinicians. This instrument is well worthy of trial before any objections are to be considered. . . . It is my hope that the findings of Harrington and Parsons will be substantiated and that the ophthalmologist will find that the new tonometer is all that it appears to be—superior not only to other impression tonometers but to other types of tonometers.

Mention has been made of the Souter[23] tonometer. This is a well balanced spring instrument about the size and weight of a fountain pen, simple in construction and in method of application. It gives direct readings which are taken with the patient upright. Lloyd[2] has stated that the accuracy of this instrument is not to be compared with that of the Schiøtz and the McLean, but that if readings with the patient upright were desired his choice of instrument would be the Souter rather than the Bailliart. Harrington declared that the spring element would tend to introduce inevitable inaccuracies in the readings of a tonometer of this type. Verhoeff,[24] on the other hand, has relied on the Souter tonometer for over twenty years. He has stated that in the use of this instrument all variables pertaining to the eye other than the actual intraocular pressure become relatively unimportant, that this tonometer is more precise than the Schiøtz and that it gives a more accurate indication of the actual intraocular pressure in the living eye than is usually obtained even by laboratory methods. Verhoeff declared that, in addition to its greater accuracy, the Souter tonometer has two important advantages over the Schiøtz instrument: It is less dangerous to use from the standpoint of possible corneal abrasion and it is less objectionable to the patient. The one disadvantage, he stated, rests in the difficulty of determining the amount of indentation of the cornea at which the instrument is to be read, but once one has acquired this ability by practice there is less difficulty in making readings with this instrument than with the Schiøtz.

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23. Souter, W. N.: Simple Tonometer for Clinical Use, Ophth. Rec. **25**:80, 1916.
24. Verhoeff, F. H.: The Souter Tonometer, Am. J. Ophth. **20**:720, 1937.

When Ferree and Rand,[25] in 1931, introduced and described an apparatus which they had constructed as a checking standard for tonometers, they called attention to a definite shortcoming in connection with the clinical test of tonometry:

> In a survey of the literature it is amazing to see how much work and ingenuity have been expended on the devising of tonometers of different kinds and how little attention has been given to means for checking them for constancy and uniformity of performance. . . . There seems to be no type of test or measurement in the practice of ophthalmology more acutely in need of standardization than tonometry.

In 1942 Reese,[26] with the evidence of his own experiences and those of Friedenwald and Harrington and Parsons at hand, gave reemphasis to this situation, saying:

> There is a tendency among ophthalmologists to accept the readings of a tonometer without questioning the instrument's accuracy, but tonometers should not be relied upon implicitly unless they have been checked and their accuracy or margin of error established. . . .
>
> The Schiötz tonometer is employed more than any other, and its mechanical accuracy is of the very first importance, since the nomogram, or chart by means of which the readings are converted into millimeters of mercury, is based on an instrument designed by Schiötz and having definite and precise specifications. When we buy a Schiötz tonometer we are paying the price of, and expect to secure, an instrument of precision. Unfortunately, such is usually not the case.

Reese stated that checking stations for Schiøtz tonometers were established in Tübingen by Arnold, in Oslo by Schiøtz and in Berlin by Comberg in 1923, 1925 and 1928, respectively, but that we did not have such a checking station in this country until 1935. At that time, at the suggestion of Dr. Mark J. Schoenberg, a station was established at the Hermann Knapp Memorial Eye Hospital, New York, by Dr. Adolph Posner. During the several years this station functioned, Dr. Posner checked about two hundred tonometers and found about one third of them to be incorrect; the majority of these were of American make. Reese stated the belief that checking stations for tonometers should be conducted in this country and that ophthalmologists should feel morally compelled to have their instruments certified at such stations.[27]

There is no gainsaying the fact that within recent years the efforts of Friedenwald and of Harrington and Parsons have resulted in valuable contributions toward the long sought goal of standardization. As further evidence of the recognition of this need, a committee known as the Committee on Standardization of Tonometers, consisting of Drs. Adler, Friedenwald, Posner, Gifford, Kronfeld, Gradle and Harrington, was recently appointed[28] on recommendation of the Council of the American Academy of Ophthalmology and Otolaryngology.

99 Lafayette Avenue.

25. Ferree, C. E., and Rand, G.: A Checking Standard for Tonometers, Arch. Ophth. **6**:689 (Nov.) 1931.

26. Reese, A. B.: Standardizing and Checking Tonometers, Am. J. Ophth. **25**:478, 1942.

27. A station for checking the accuracy of original Schiøtz tonometers is maintained at the headquarters of the National Society for the Prevention of Blindness, 1790 Broadway, New York. In reply to an inquiry, the society reported that at the present time this checking station is the only one in the United States.

28. Tr. Am. Acad. Ophth. **47**:138, 1942.

Obituaries

JOHN MILTON GRISCOM, M.D.
1881-1943

J. Milton Griscom was born in Salem, N. J., Feb. 23, 1881 and died June 5, 1943, at the Pennsylvania Hospital, in Philadelphia. While he had endured with fortitude much suffering from a sinus and allergic condition for seven years, there had been no evidence or suggestion of cardiovascular disease. His first

JOHN MILTON GRISCOM, M.D.
1881-1943

attack came suddenly, on June 3. He was taken home and two days later was removed to the Pennsylvania Hospital, where he died that evening of coronary thrombosis. He was the son of Walter Davis Griscom and Mary McIlvaine Bessett, members of the Society of Friends.

His early education was acquired at Friends' School and the Swarthmore Preparatory School. He entered Swarthmore College and graduated with the degree of Bachelor of Science in 1902. His years at Swarthmore were always

pleasant memories. His jovial manner and musical talent as a member of the college glee club attracted many friends, who were loyal throughout the years. It was here he met Mary Lippincott, whom he married in October 1915.

Within a few weeks after he received his degree at Swarthmore, his father was appointed superintendent at Wills Hospital. This fortunate coincidence was the deciding factor in young Griscom's medical career. He was admitted to the medical school of the University of Pennsylvania and graduated with the degree of Doctor of Medicine in 1906. He then served one year as intern at the Germantown Hospital, in Philadelphia.

His career at Wills Hospital began as intern in 1908 and continued for a span of thirty-five years. The staff at Wills during this period included such eminent men as Oliver, Posey, Zentmayer, Ziegler and Sweet, all members of the American Ophthalmological Society. From these men he gained not only a thorough knowledge of ophthalmic practice and surgery, but a strict rule of ethics, which he practiced to the end. He was assistant surgeon in 1910, attending surgeon in 1926 and executive surgeon for the past several years.

In 1914 he was elected to the American Ophthalmological Society. His interest and devotion to this society was attested by his work: From 1929 to 1933 he served as editor of the *Transactions;* from 1933 to 1937, as secretary-treasurer, and from 1938 to 1943 as member of council.

When the Graduate School of Medicine of the University of Pennsylvania was organized, Dr. Griscom was made associate professor, and shortly after professor, of ophthalmology. He served as consultant to the Jeanes Hospital, Philadelphia, and to the Burlington County Hospital, at Mt. Holly, N. J. He was a member of the American Medical Association, the Philadelphia County Medical Society, the Medical Society of the State of Pennsylvania and the American Academy of Ophthalmology and Otolaryngology.

He was elected fellow of the College of Physicians of Philadelphia in 1912. He served as clerk of the section on ophthalmology of this society and later as chairman. During his early years he presented a number of interesting cases and papers before this society. Dr. Griscom was not a frequent writer, but he contributed freely of his time in lectures before the ophthalmic section of the Philadelphia County Medical Society. He was a member of Phi Kappa Psi fraternity and the Union League.

He is survived by his wife; a daughter, Mary Lippincott Jr., and a sister, Mrs. J. Passmore Elkinton. Since his marriage he had lived in Moorestown, N. J., where he took an active interest in the Friends' School. He was devoted to his family and enjoyed the comfort of his delightful home above all. His vacation was usually spent at Buck Hill in the Pocono Mountains, where for many years he enjoyed and played an excellent game of golf.

It was my privilege to know Dr. Griscom intimately since our initial meeting at a meeting of the American Ophthalmological Society at New London, Conn., in 1915. He was a gentleman, quiet in manner, unassuming and conscientious, and gave the best that was in him to the service of his patients.

CHARLES R. HEED.

LESLIE J. PATON, M.B., F.R.C.S.

1872-1943

Leslie Paton died in London on May 15, after a long illness. Born in Edinburgh, Scotland, in 1872, the second son of James Paton, F.L.S., curator of picture galleries and museums, he received his early education in the Glasgow high school and the University of Glasgow and at Caius College, Cambridge University. He graduated from the medical school of St. Mary's Hospital, London, with the degree of Bachelor of Medicine, and received the qualification of Fellow of the Royal College of Surgeons in 1902. Paton began his ophthalmologic studies as assistant to Henry Juler at St. Mary's Hospital and succeeded him in the post of ophthalmic surgeon in 1911. He also worked with Marcus Gunn, both at Moorfields and at the National Hospital, Queen Square, and was appointed Gunn's successor as ophthalmologist to the National Hospital in 1907, where he had the privilege of following the teachings of that great neurologist Hughlings Jackson. Paton had the double distinction of being chosen president both of the Neurological Section of the Royal Society of Medicine and of the Ophthalmological Society of the United Kingdom. As suggested by his training, one of his principal interests was in ophthalmic neurology, and his chief, and most important, contributions to ophthalmic literature were on neurologic subjects, such as "The Pathology of Papilloedema," in collaboration with Gordon Holmes; "Optic Atrophy in Tabes," and his Mackenzie Memorial Lecture in 1937, "Demyelinating Diseases accompanied by Optic Neuritis." His administrative capacity had full play in the important work that he performed as treasurer of the International Ophthalmological Council and as managing director of the *British Journal of Ophthalmology*, when he conducted the affairs of that journal to safety in the difficult years after the first world war.

Leslie Paton was tall and handsome, a kindly and strong personality, thorough and earnest in his work, and his death is a great loss to ophthalmology and to his many friends. As Dr. Wilfred Harris [1] stated:

> Leslie Paton made few, if any, enemies, though he was highly successful in practice. His brain was brilliant and analytical, but he took his time in making up his mind. Though unknown personally to the present generation of students at St. Mary's he will be long remembered by his friends and generations of past students for his teaching, his balanced mind, and his cheery disposition.

ARNOLD KNAPP

I was resident medical officer at the National Hospital from 1907 to 1910 inclusive, nearly four years, during which time Mr. Marcus Gunn and Mr. Paton were the attending ophthalmologists of the hospital. Mr. Paton made rounds once or twice every week and taught the value of careful study and the importance of small things. His interest in our first case of ipsilateral optic nerve atrophy and contralateral papilledema, occurring as a result of a meningioma of the subfrontal region, was great, and his encouragement led me to further studies on this and other ophthalmic problems occurring in neurologic medicine.

Mr. and Mrs. Paton were exceedingly kind to me, almost giving me the run of their house in Harley Street. Both of them remained as Scottish as the "Kyles of Bute," and their kind lives were illuminated always by their cheery hearts and pawkish humor.

1. Leslie J. Paton, Obituary, Brit. M. J. **1**:741 (June 12) 1943.

I have seen Paton practically every year since my acquaintance with him and had the pleasure of a long letter from him last year, after his daughter and son-in-law had paid us a week's visit in Bar Harbor.

By his death the neurology of the eye has lost perhaps its most distinguished exponent, and I have lost one of my closest friends.

FOSTER KENNEDY

LESLIE J. PATON, M.B.
1872-1943

In the death of Leslie Paton, his family and friends have suffered an irreparable loss and ophthalmology has lost one of its outstanding members. His keen mind and tenacious memory gave him a fund of information that was most unusual.

Every one who visited Queen Square while he was on duty could not but be impressed with the sincerity of his work and the meticulous and broad manner in which he considered his cases. His ability as an organizer is well shown in the part he played in the reorganization of the International Congress of Ophthalmology after World War I.

He was tireless in his efforts, and all who were associated with him appreciated fully his sincerity and fairness in his methods of procedure. His sterling honesty, his calm demeanor and his grasp of every situation that presented itself were greatly admired by all who were associated with him.

It was a great privilege to know Leslie Paton and to become acquainted with his quiet, unruffled nature and his wise counsel. His death leaves a gap which cannot be filled, but all are richer for the inspiration and example of his life.

WALTER R. PARKER

News and Notes

EDITED BY DR. W. L. BENEDICT

SPECIAL NEWS

Leslie Dana Gold Medal Award.—Dr. Walter B. Lancaster, of Boston, has been awarded the Leslie Dana Gold Medal. This medal is awarded annually for outstanding achievements in the prevention of blindness and in the conservation of vision. The selection of Dr. Lancaster for this honor was made by the St. Louis Society for the Blind, through which the medal is offered by Mr. Leslie Dana, of St. Louis, on recommendation of the Association for Research in Ophthalmology.

The conditions of the Leslie Dana Medal award set forth that it is to be made for "long meritorious service in the conservation of vision in the prevention and cure of diseases dangerous to eyesight; research and instruction in ophthalmology and allied subjects; social service for the control of eye diseases, and special discoveries in the domain of general science or medicine of exceptional importance in conservation of vision."

Notice

If the demand is sufficient, the American Medical Association Press will reprint the series of lectures by Prof. Alfred Bielschowsky on Motor Anomalies of the Eyes, which was published in the December 1934 issue of the ARCHIVES, page 805, and in the January, April and May issues of 1935, pages 33, 569 and 751.

A form is printed below for the convenience of those who wish to order the reprint. The price will depend on the number of reprints ordered, but it will not exceed $2.50.

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ADIE'S SYNDROME

To the Editor:—Dr. Rooks's article "Adie's Syndrome," in the June issue (ARCH. OPHTH. **29:** 936, 1943) suggests that all cases of this syndrome "be brought to the attention of the medical profession with as much publicity as possible." I am in complete agreement with this idea, and I assume that if this is the purpose of the report, the cases which are described should be typical of the syndrome; should not be unusual and misleading varieties; should be carefully, completely and fully worked up, and should leave no doubt in the reader's mind as to just what the Adie syndrome is. I am aware that in conclusion the author states:

> . . . these 2 cases are offered for their clinical and scientific importance, with the hope that further interest in the study of Adie's syndrome may be stimulated, but my limited knowledge of the fundamental nature of this disorder warrants no specific conclusions.

The first case presented was that of a man aged 44. A definite history of head injury was noted eighteen months and inequality of the pupils eight weeks prior to examination.

> The left pupil was 5.5 mm. in diameter and firmly fixed. . . . Prolonged exposure to bright artificial light or to direct sunlight failed to produce any change whatever in the size and shape of the left pupil, nor did its size and shape change after the patient remained in a completely dark room for thirty minutes. . . . The left pupil remained dilated and absolutely inactive to light and accommodation.

These many statements require elucidation, for the findings are atypical of Adie's syndrome and serve to confuse, rather than to clarify, the clinical picture. It has been pointed out (and by men to whom the author refers, viz., Adie, Saenger, Strasburger and Behr) that the most consistent, the most easily recognized and for diagnostic purposes the essential feature of this syndrome is the behavior during and after convergence. The author states that the myotonic pupil may be "incompletely or entirely indifferent to convergence and to accommodation." Such a response is an exception to the rule, and in Alajouanine and Morax's review of 100 cases this was rarely reported!

Dimmer, Behr and Lowenstein stated (and it is now established) that the second most characteristic feature of the syndrome is dilatation of the Adie pupil when the patient remains in darkness for one hour and that when the patient comes out into the light a sluggish contraction of the pupil occurs after an interval.

Most cases of the Adie syndrome occur in young persons, and most of the patients are highly emotional—case 1 fulfils neither of these requirements.

In the second case outstanding discrepancies are the patient's age, the absence of the characteristic delayed reaction to convergence and the pronounced pupillary irregularity. Again, a question arises as to the bilaterality of the syndrome inasmuch as the left pupil was 3.5 by 2.5 mm. and reacted "well both to light and in accommodation." Since the Adie syndrome is one of "mid mydriasis," with no normal reaction to light or to accommodation, this statement is contested, especially since Behr asserted that "a pupil not more than 3 mm. in diameter is never an Adie pupil."

Many other tests and features might have been used to clarify the syndrome, such as tests of accommodation, fatigue of accommodation, superimposition of convergence on accommodation and its effect on size of the pupils. The reaction to various drugs, such as mecholyl, strychnine, cocaine and physostigmine, as pointed out by Cogan, Adler, Kyrielies and Lowenstein, might add certainty to the diagnosis. A history of vasomotor changes and emotional disturbance and a study of other ocular reflexes might also be considered.

I believe every case of this syndrome should be reported and completely worked up, and if such reports are for the benefit of the "medical profession," then typical rather than atypical forms of the disease should be reviewed.

EMANUEL ROSEN, Captain, M.C., A.U.S.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

FUNCTIONAL REPRESENTATION IN OCULOMOTOR AND TROCHLEAR NUCLEI. M. B. BENDER and E. A. WEINSTEIN, Arch. Neurol. & Psychiat. **49:** 98 (Jan.) 1943.

The article deals with localization of function in the cell masses of the oculomotor nucleus. Electric stimulations and lesions were made in the oculomotor and trochlear nuclei of monkeys. The experiments indicate that individual ocular muscles are functionally represented within the ipsilateral oculomotor nucleus, while the superior oblique muscle is governed by the contralateral trochlear nucleus. The dorsoventral and rostrocaudal arrangement of functional representation of the ocular muscles is as follows: (1) sphincter pupillae; (2) inferior rectus; (3) ciliary (?); (4) inferior oblique (?); (5) internal rectus; (6) superior rectus; (7) levator palpebrarum; (8) superior oblique (contralateral). R. IRVINE.

Conjunctiva

MEMBRANOUS INFLAMMATION OF OROPHARYNX, NOSE AND CONJUNCTIVA DUE TO SULPHATHIAZOLE ADMINISTRATION. I. M. SCHNEE, Brit. M. J. **1:** 506 (April 24) 1943.

The following case is reported as evidence of an unusual type of toxic reaction from therapy with a sulfonamide compound. The reaction consisted in inflammation involving the conjunctiva, nose, pharynx, mouth and larynx, with the development of a membrane. There was rapid recovery on discontinuance of the chemotherapy.

The patient, a soldier 28 years of age, had been treated with sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) for an infection of the upper respiratory tract. After he had received about 18 Gm. the symptoms seemed to be relieved and treatment was stopped. On the fifth day of chemotherapy the eyes became inflamed and a discharge was present; then the mouth became very sore and the lips cracked. The fever recurred, and the patient was sent to the hospital. The lids were swollen, with a smooth, thick, well formed gray membrane which left no bleeding surface when peeled off but reformed within a few hours. The cornea was not involved. A similar condition was found in the mouth and throat. Smears and cultures were negative except for staphylococci. On the patient's relapse, sulfathiazole treatment was renewed. When the condition of the eyes and throat became worse, at the suggestion of Lieut. Col. Derrick T. Vail use of the drug was discontinued. The patient rapidly became well.

ARNOLD KNAPP.

Congenital Anomalies

ANOMALOUS PROCESSES OF CILIARY TYPE ON POSTERIOR SURFACE OF IRIS. S. GARTNER, Am. J. Ophth. **25:** 854 (July) 1942.

Gartner gives the following summary:

"Twenty-six cases are reported of anomalous processes on the posterior surface of the iris. They closely resemble the ciliary processes, from which they are separated by normal iris tissue. The anomalous processes are fetal remains of

tissue which usually disappear by the time of birth. Their presence can assume practical importance in complicating an iridectomy, in as much as they may cause increased hemorrhage and postoperative reaction."

W. S. REESE.

DISPLACEMENT OF CILIARY PROCESSES TO POSTERIOR SURFACE OF IRIS. S. GARTNER, Am. J. Ophth **25:** 858 (July) 1942.

Gartner gives the following summary:

"Partial displacement of ciliary processes to the posterior surface of the iris is a common variation from the normal. This is explained by the embryology of the ciliary processes. Its importance in iridectomy is discussed."

W. S. REESE.

Cornea and Sclera

CASE OF BILATERAL INTERSTITIAL KERATITIS LEADING TO BLINDNESS. J. D. J. FREEMAN, Brit. J. Ophth. **27:** 104 (March) 1943.

The patient was a stoker aged 19. The morning after a "blitz" during which, without incurring injury, he noted a "very bright flash," the left eye became inflamed. Examination showed beginning interstitial keratitis. In about six weeks the eye was shrunken and blind. There was panophthalmitis, and the cornea had evidently perforated. The eye was excised. About eleven weeks from the time the left eye became inflamed, interstitial keratitis developed in the right eye. The outcome was an opaque cornea, a soft eye and vision equal to light perception. The Wassermann reaction of the blood was strongly positive. The treatment was directed to the syphilitic infection, and later a course of tuberculin was given. The author describes the case as one of very severe congenital syphilitic infection of the eyes starting as interstitial keratitis and going on to bilateral gummatous infiltration of the uveal tract and loss of both eyes.

W. ZENTMAYER.

CONTACT LENS IN CASES OF NEUROPARALYTIC KERATITIS. M. KLEIN, Brit. J. Ophth. **27:** 221 (May) 1943.

After an operation for an acoustic tumor on the right side the patient had complete palsy of the right facial nerve and trigeminal anesthesia. Neuroparalytic keratitis developed, for which a tarsorraphy was done. After a year, though the lids were still sutured, the condition of the eye seemed hopeless. In August 1942 the patient was fitted with a contact lens. Rapid improvement followed, and within two or three weeks the eye became white and the cornea cleared considerably except at the central area, where a dense scar remained. In November 1942 the patient broke the contact lens; within a few hours the eye became inflamed and the following day the infiltration was extensive. A new glass was supplied, and within a week the eye quieted down.

In a second case, in which neuroparalytic keratitis developed, with involvement of the cornea, after an alcoholic injection for trigeminal neuralgia on the right side, a contact lens gave an equally good result.

W. ZENTMAYER.

General Pathology

PATHOLOGY OF CHOLESTERIN AND FAT DEPOSITION IN MUSTARD GAS INJURIES OF THE CORNEA. I. MANN and B. D. PULLINGER, Brit. J. Ophth. **26:** 503 (Nov.) 1942.

In a previous paper Mann and Pullinger showed that, of experimentally produced mustard gas lesions of the eyeball, central corneal lesions healed without vascularization of the cornea and did not lead to deposition of cholesterol or fat in the scars. It is therefore considered that the essential factor in this

degeneration is previous vascularization of the cornea. In some cases the vascularization was superficial; this type was never followed by deposition of fat or cholesterol. In other cases the vessels penetrated the substantia propria, with separation of the lamellas by edema fluid ahead of the invading vessels. These deeper vessels showed extremely bizarre shapes and irregular caliber and extended in the form of tips or pointed ends, in which no movement of blood could be seen. The pattern varied from day to day, always extending farther into the cornea and always accompanied by edema. The arrangement of bulbous ends and right-angled branches suggested so-called Bowman canals. As the vessels retrogressed, in a relatively small number of cases a deposit of fat and cholesterol was formed beyond their disappearing tips. It was noticed that the series of eyes which received treatment showed a much higher percentage of degenerative scars than did those in which the lesion was allowed to run its usual self-limited course. The lipoids occur in two forms, as exceedingly fine droplets, like an emulsion, bathing the fibrils of the substantia propria and as larger intracellular globules and crystals having the characteristics of esters of cholesterol.

W. ZENTMAYER.

Glaucoma

OPERATION FOR CONGENITAL GLAUCOMA. O. BARKAN, Am. J. Ophth. **25:** 552 (May) 1942.

Barkan presents the following summary and conclusions:

"In this paper the results of operations on 17 eyes with congenital glaucoma are reported. Pressure was normalized in 16 of the 17 eyes; visual function was maintained in 14. Of the three blind eyes brought to operation, vision was lost in two before surgery was instituted. In only one case did operation fail to normalize the pressure in time to save vision. Sight was preserved in the fellow eyes of these three infants so that they were saved from blindness. The average age at time of operation was three months. From one to five years have elapsed since the operations were performed. On the basis of the successful results and of pre- and post-operative gonioscopy, certain conclusions are arrived at in regard to the underlying pathology and to the mode of action of the operation. It appears that the majority, if not all, binocular cases of congenital glaucoma are due to a developmental obstruction in the excretory angle which, when removed, permits outflow of aqueous into the canal of Schlemm. The canal is found to be present and ready to function in the large majority of cases in which the diagnosis is made at a sufficiently early age, before prolonged distention of the eyeball has obliterated the canal. Early diagnosis and early operation are therefore urgently indicated. The operation gives a high promise of permanent success with a remarkable degree of safety and without disfigurement."

W. S. REESE.

THE PRACTICAL APPLICATIONS OF GONIOSCOPY TO GLAUCOMA SURGERY. H. S. SUGAR, Am. J. Ophth. **25:** 663 (June) 1942.

Sugar discusses the practical aspects of gonioscopy in operations for glaucoma, his classification of glaucoma and the clinical picture of acute glaucoma. He briefly reviews the different operations for glaucoma and their indications and efficacy, especially from gonioscopic observations.

W. S. REESE.

GLAUCOMA. T. H. BUTLER, Brit. J. Ophth. **27:** 116 (March) 1943.

This is a lecture on glaucoma given during the course for the diploma of ophthalmology at the University of Oxford. It is of sufficient interest to be noted, as many of the views expressed are heterodox but debatable.

W. ZENTMAYER.

Injuries

REMOVAL OF SMALL MAGNETIC FOREIGN BODIES FROM THE EYE EIGHTEEN MONTHS AFTER THE DATE OF INJURY. H. NEAME, Brit. J. Ophth. **27:** 226 (May) 1943.

The patient was admitted to the hospital because powdered chalk had entered his eye. There was a history of injury to the eye eighteen months before while he was knocking off the head of a screw with a hammer and chisel. There were a corneal scar to the nasal side and a dark rust-colored nodule on the iris behind the scar. The foreign body was freed from the iris and then removed through a keratome section.

The case is reported as an indication that minute magnetic foreign bodies may be retained for a considerable period without setting up appreciable siderosis. The article is illustrated.

W. ZENTMAYER.

A CASE OF MULTIPLE INTRA-OCULAR FOREIGN BODIES. R. A. D. CRAWFORD, Brit. J. Ophth. **27:** 227 (May) 1943.

The patient was stamping out steel airplane parts on a hand press when he felt a sting in the right eye. There was a small perforation wound above the center of the cornea, with a track of a foreign body through the lens. In the vitreous there were a fine glistening foreign body about 3 mm. long lying anterio-posteriorly with one end in the center of the disk, a smaller foreign body at 6 o'clock at the bottom of the vitreous and two minute foreign bodies in the vitreous near the macula. The largest foreign body was found weakly magnetic when the Haab magnet was used under direct ophthalmoscopic observation. The magnet was used once or twice a day for two weeks, at the end of which the body came clear of the disk. After a rest of ten days, the magnet was applied on two successive days; the foreign body came into the anterior chamber but returned through the pupil when the current was turned off. It was removed through a keratome section by hand and giant magnets.

It is suggested that less damage was inflicted than would have resulted from employment of the posterior route.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

SURGICAL TREATMENT OF VASCULAR DISEASES ALTERING THE FUNCTION OF THE EYES. A. W. ADSON, Am. J. Ophth. **25:** 824 (July) 1942.

This article deals mainly with pulsating exophthalmos, but aneurysms of the chiasmal region and angiomatous tumors are briefly discussed, particularly with regard to their surgical management.

W. S. REESE.

DIABETES INSIPIDUS AND OTHER UNUSUAL COMPLICATIONS OF ACUTE PURULENT SINUSITIS: CLINICOPATHOLOGIC STUDY OF A CASE. J. C. YASKIN, F. H. LEWEY and G. SCHWARZ, Arch. Neurol. & Psychiat. **48:** 119 (July) 1942.

The authors report the case of a 21 year old man who showed osteitis of the right lesser wing of the sphenoid bone as a complication of purulent sinusitis. The inflammation spread across the sella turcica and gave rise to changes in the optic nerves. After operation on the sphenoid sinus there developed successively subarachnoid hemorrhage, basilar meningitis and involvement of the floor of the third ventricle, with consequent diabetes insipidus and changes in the cerebrum, which eventually led to generalized convulsions, high fever and death.

Optic neuritis is not a rare complication of disease of the sphenoid and ethmoid sinuses, although there are observers who believe that retrobulbar neuritis is rarely, if ever, due to disease of the paranasal sinuses. In this case it was a striking feature in that the optic nerve atrophy developed during the period of observation in the hospital, in the course of a few days, undoubtedly as a result of the local inflammatory process around the optic chiasm and optic nerves.

R. IRVINE.

ORBITAL EMPHYSEMA SIMULATING CELLULITIS. C. L. C. O'MALLEY, Brit. J. Ophth. **27**: 222 (May) 1943.

A case is described of severe proptosis, endangering the eye, which followed a stab wound made by a football player's finger. A slight hemorrhage from the eye and left nostril was followed by closure of the eye. Proptosis began on the third day. The cornea became opaque and sloughed at the exposed area. There was marked chemosis, and the eyeball was stony hard. The symptoms were indistinguishable from those seen in orbital cellulitis. An evacuation operation determined that the proptosis was due to gaseous accumulation in the orbit. The eye, which looked a total loss, made an almost complete recovery.

To the author the most likely explanation is that the finger made a valvelike opening in the orbital plate of the ethmoid bone, which allowed air under pressure to enter the orbit.

W. ZENTMAYER.

Parasites

REMOVAL OF FILARIA WORM FROM EYE. M. C. T. REILLY, Brit. M. J. **1**: 103 (Jan. 23) 1943.

A middle-aged officer who had spent considerable time in Nigeria requested that a filaria be taken out of his right eye. There was marked injection of the conjunctiva, and a worm could easily be seen wriggling underneath toward the outer canthus. By the time the patient reached the hospital the worm had disappeared, but in half an hour it appeared again in the fornix of the lower sac. The eye was cocainized, and after a small incision was made the worm was seized with a forceps. Recovery was uneventful. The worm measured 32 mm. The patient had been in England four years since his service in Nigeria. While there he twice had worms removed from his right eye and once suffered from typical "Calabar swellings" on his wrist. Otherwise his health had been good.

ARNOLD KNAPP.

FILARIA WORM IN THE EYE. E. A. CHARTRES, Brit. M. J. **1**: 269 (Feb. 27) 1943.

The writer says that forty years ago, when he was in northeast India, worms in the eyes were common in horses. It was necessary to immobilize the horse, cocainize the eye and wait, while kneeling on the horse's neck, for the worm to appear in the anterior chamber; then a knife was plunged slantwise into the anterior chamber and the worm extracted. The worm frequently measured from 1½ to 2 inches (4 to 5 cm.). The eye was always inflamed and the cornea opaque with a milky fluid, but cleared up after a few days. The condition if not discovered will destroy the eye.

In Nigeria, filarias are common, and the writer himself, while there, was a sufferer from a worm in the eye, which sometimes caused him inconvenience. He had frequently seen it wriggling in his eye and under the skin of the temple. The trouble lasted six years.

The worms are also common in antelopes.

ARNOLD KNAPP.

Retina and Optic Nerve

MARGINAL HAEMORRHAGE ON THE DISC: PARTIAL CROSS TEARING OF THE OPTIC NERVE; CLINICAL AND HISTOLOGICAL FINDINGS. A. LOEWENSTEIN, Brit. J. Ophth. **27:** 208 (May) 1943.

A man aged 38 was injured by a nail which fell on the right eye from a considerable height. A perforating wound was found in the sclera at 4 o'clock, 2 mm. from the limbus. There were hyphemia and marked hypotonia. Visual acuity equaled seeing of fingers at 0.5 meter. On clearing of the media a clear arcuate band was visible at the temporal margin of the disk from 11 to 7 o'clock. The breadth was approximately that of a central vein. Later three fine radially striated hemorrhages were present about the disk. The field of vision showed concentric contraction. Judging by the histologic structure of the normal retina, the author concludes that the red crescent was caused by the bleeding within the sheaths of the optic nerve, subdural or subarachnoid, with infiltration of Elschnig's border tissue.

Studying the histologic material of excised eyes at the Tennent Institute, Loewenstein found 3 instances of partial cross rupture of the optic nerve at the insertion of Bruch's membrane among a relatively small number of eyes examined. The tears ocurred in three different conditions: trauma, hemorrhagic glaucoma and expulsive hemorrhage. All 3 occurred exactly at the insertion of Bruch's membrane. They were produced by instantaneous, probably arterial, subretinal bleeding.

The article is illustrated.

W. ZENTMAYER.

Trachoma

TRACHOMA: ITS PROPHYLAXIS IN ARGENTINA. J. A. SENA, Semana méd. **49:** 1520 (Dec. 24) 1942.

According to Sena, about 15 per cent of the population of the world have trachoma. No country in the world is entirely free from it. It is a disease of poverty, promiscuity, unhygienic living conditions and poor food. The diagnosis is difficult. Numerous diagnostic aids have been suggested, such as the complement deviation test, examination for eosinophilia, the phytotoxin test of Macht, the lysozyme titer in the lacrimal fluid, Abderhalden's reaction and the Weil-Felix reaction, but none of these have fulfilled the hopes placed in them. The diagnosis is chiefly clinical and is based on the presence of follicles in the tarsal portion of the conjunctiva, the presence of pannus, the appearance of cicatrices and palpebral and tarsal alterations such as entropion and trichiasis. The treatment is local as well as general. Measures which aim at improvement in the general health should not be neglected. For the incipient forms of trachoma in children mild eyewashes of zinc or copper sulfate are used. The therapeutic armamentarium against the more severe forms includes copper sulfate, silver nitrate, chaulmoogra oil and mercuric cyanide. Physical methods include cauterization and diathermic coagulation. Mechanical methods, such as massage, brushing and curettage, aim at rapid destruction of the trachomatous follicles. Biologic methods occasionally used are autoserotherapy and vaccine treatment. Cicatricial trachoma requires surgical treatment. Opinions are divided as to the efficacy of the sulfonamide compounds. They are of great value in the complicated forms. The disease is most prevalent and is practically endemic in the northern and subtropical parts of Argentina, where it is favored by climatic, social, sanitary and economic conditions. It is not autochthonous for these regions but was brought in by immigrants, chiefly from the Mediterranean countries. Trachoma of children is best combated in the schools. This has been done in recent years with the aid of visiting nurses. Ophthalmologic dispensaries should provide free treatment for adults. Since 1936 the national department of health has provided antitrachoma

drugs for the dispensaries and schools. Mobile medical ambulances would be helpful in bringing treatment to patients in the rural regions. Popular publications, illustrations and moving pictures are valuable in disseminating information regarding the prophylaxis of the disease. J. A. M. A. (W. ZENTMAYER).

Tumors

INTRACRANIAL DERMOID AND EPIDERMOID TUMORS. J. MARTIN and L. DAVIS, Arch. Neurol. & Psychiat. **49:** 56 (Jan.) 1943.

The definition of epidermoid and dermoid tumors is interesting because of their frequent occurrence about the eye. This article deals with intracranial dermoids and is summarized as follows:

"1. An epidermoid tumor is a benign neoplasm, arising from an embryonic inclusion, or nest of cells. The tumor consists of epidermal cells in various stages of disintegration, together with variable proportions of crystalline cholesterol: This tumor, containing cellular components only from the epidermis, should not be confused with cholesteatoma, which is a product of chronic inflammation.

"2. A dermoid tumor is a benign neoplasm, arising from an embryonic inclusion or nest of cells, the contents of which may represent part or all of the derivatives of the ectoderm.

"3. A teratoid tumor contains representatives from the ectoderm and either one of the other two germinal layers. This tumor bridges the gap between the true dermoid and the true teratoma, which contains components of all three germinal layers.

"In most large series of verified tumors epidermoids are four times as common as dermoids, but in our own group of over 700 verified intracranial tumors this proportion is reversed.

"Epidermoid and dermoid tumors may become manifest at any age, and they do not show any particular relationship to sex, trauma or other incidents in the medical history.

"The dermoid occurs at many different intracranial sites, but is frequently attached to the dura mater, is commonly seen in the midline and most often lies below the tentorium cerebelli. The epidermoid is practically always located in the cerebellopontile angle.

"Frequently roentgenograms reveal local erosion of bone in the skull, with an area of sclerotic bone immediately surrounding the eroded area, and within the eroded space flecks of calcium may be visible. Such lesions should not be regarded as infallible evidence of the presence of an intracranial epidermoid or dermoid tumor, since other intracranial tumors may produce the same roentgenologic changes.

"These tumors may be operated on successfully. In all 5 of our cases the patient has completely recovered from the operation. Two patients who were not immediately relieved of all their neurologic symptoms were greatly improved immediately after operation and are still improving." R. IRVINE.

Society Transactions

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

RALPH I. LLOYD, M.D., *President*

W. L. BENEDICT, M.D., *Secretary*

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Embryologic Basis of Extrapapillary Coloboma and Conus. President's Address. DR. RALPH I. LLOYD, Brooklyn.

Symposium on Chemotherapy.

OPHTHALMOLOGY: JOHN G. BELLOWS, M.D., Chicago.

OTOLARYNGOLOGY: CHARLES T. PORTER, M.D., Boston.

INTERNAL MEDICINE: JOHN A. KOLMER, M.D., Philadelphia (by invitation).

Congenital Ptosis: Factors Which Indicate Necessity for Varied Surgical Procedures. DR. EDMUND B. SPAETH, Philadelphia.

Pronounced differences in the following factors are characteristic of congenital ptosis: (*a*) the degree of involvement of the levator muscle; (*b*) the unilaterality or bilaterality of the condition; (*c*) the presence and degree of accompanying involvement of the superior rectus muscle, and (*d*) the more extensive complications, such as the jaw-winking reflex and the retraction syndrome. The age and the conditions associated with age at the time the patient appears for operation are also factors.

The variability of these factors indicates conclusively that no single operative procedure can correct more than one set of conditions. The fact that so many operations have been devised for ptosis is proof of this, and not an indication of general dissatisfaction with all forms of surgical treatment of ptosis, as has repeatedly been stated. The mechanical difficulties connected with elevation of a ptotic lid cannot be solved by any single operation. Success means that the patient must have a good functioning lid; i. e., it must lift satisfactorily and close normally, and in downward gaze the margin of the lid must follow the movement of the cornea. The winking reflex must be normal, and the patient must have a good orbitopalpebral fold. These requirements can be met most closely by careful study of each case. The surgical procedure which best meets the most important requirements in the case under consideration is the one to be used.

DISCUSSION

DR. BERNARD J. LARKIN, Indianapolis: Dr. Spaeth's able presentation has covered the subject so well that there is nothing to add. The fine organization of his material makes it easy to follow him to his conclusions. He has properly placed congenital ptosis, and this is sufficient reason for appreciative consideration of his research.

He has stressed the importance of early diagnosis and treatment, for in 37 per cent of the cases in his series factors other than epicanthus and ptosis were presented. He suggests that the use of crutch glasses after atropine refraction will assist the child in overcoming these complications, so that operation may be postponed until he is 4 or 5 years old; that not infrequently paralysis of the superior

rectus muscle is of equal importance in production of complications of ptosis which are more serious than the cosmetic blemish, and, that the anatomic classification decides the surgical procedure. He emphasizes the fact that one operative procedure will not correct all forms of ptosis.

He classifies ptosis into nine forms. On first impression it may seem that several of these types are similar, but after careful analysis one must agree with Dr. Spaeth that sufficient conditions exist for such a differentiation. Moreover, this classification becomes especially significant in analysis and presentation of the cases illustrative of the various types. For example, in cases of type 1, unilateral ptosis without involvement of the superior rectus muscles; of type 5, unilateral ptosis with weakness of both superior rectus muscles, more pronounced, however, on the homolateral eye, and of type 8, ptosis associated with the Duane retraction syndrome, shortening of the levator muscle with partial tarsectomy is the ideal procedure. In cases of type 7 recession of the internal rectus muscle may correct this retraction syndrome, as well as the ptosis; however, if this procedure is not sufficient, advancement of the levator muscle without tarsectomy will probably correct the condition. Other surgical procedures were necessary in cases of type 5, in which a recession of the overacting right inferior oblique, recession of the left inferior rectus and advancement of the left superior rectus muscle were done. This illustrates the advisability of early operation on the extraocular muscles, before the ptosis is corrected.

"Utilization of the superior rectus muscles has a narrower field of indicatability and permissibility." Dr. Spaeth seems to be the first to advocate still further narrowing of the utilization of the superior rectus muscles in cases of bilateral ptosis by use of a Shoemaker or Kirby modification of the Motais operation. In a former paper he set forth his reasons for this opinion. The postoperative care of patients undergoing operation on the superior rectus muscle is also more demanding and exacting than that of patients undergoing operation on the levator muscle.

Utilization of the occipitofrontalis muscle, when indicated, is a good procedure to follow, as the author has shown. For type 2, unilateral ptosis with involvement of the homolateral superior rectus muscle, and for type 4, bilateral ptosis with involvement of the superior rectus muscles, the Tansley-Hunt procedure is chosen, while for type 6, ptosis with more or less complete paralysis of the third nerve, and even of the sixth nerve, the Reese procedure is indicated. For type 7, ptosis with the classic jaw-winking reflex, a levator tenotomy is first performed, and later a Reese attachment of the orbicularis to the occipitofrontalis muscle is carried out.

The enlarged epicanthus which occurs in children with bilateral ptosis should not be operated on until the child is older, as the condition frequently adjusts itself when the adult facial growth is reached. However, in exceptional cases the Gifford technic is the method of procedure.

I have touched on only the points in treatment of congenital ptosis that seem to me to be most striking and pertinent.

In conclusion, I should like to repeat Dr. Spaeth's closing plea for earlier diagnosis and for as early treatment as is possible in the individual case.

Dr. Edmund B. Spaeth, Philadelphia: The portion of the paper which I could not read because of limitation of time contains a discussion, in very frank detail, of how I feel with regard to various surgical indications and their application. For instance, I do not feel that the occipitofrontalis procedure is permissible except in cases of cicatricial ptosis in adults or of cicatricial ptosis in children with complete paralysis of the levator muscle.

The jaw-winking reflex has been badly managed by most neurosurgeons. It is unfortunate that patients with this complication reach the neurosurgeons, for they become neurosurgical problems for the rest of their lives. It is a simple procedure to convert this type of ptosis into complete paralysis and then carry on from there by means of Reese's method of attachment of fibers and cicatricial bands from the orbicularis to the occipitofrontalis muscle.

Management of Iridocyclitic Secondary Glaucoma. Dr. F. Bruce Fralick and Dr. James H. Cooper and Dr. Richard Armstrong (by invitation), Ann Arbor, Mich.

A survey of 1,282 consecutive cases of iridocyclitis revealed that in 261, or 22 per cent, secondary glaucoma was present or developed while the patient was under observation. The ratio of females to males with glaucoma was approximately 2:1. The highest incidence fell in the sixth decade of life; the mean age was 53 years; the youngest patient was 20 years and the oldest 76 years of age.

The cause of the iridocyclitis associated with glaucoma was not definitely proved in any instance, but in over one half of the cases, a definite focus of infection was found.

Of the 261 eyes that had secondary glaucoma, 69 were treated medically as a primary procedure. This type of treatment was carried out over a sufficient period to make possible evaluation of the results. Administration of miotic drugs was tried in 21 eyes with more or less quiescent iridocyclitis. However, the tension was reduced to normal in only 6 eyes. Homatropine or atropine was used in 46 eyes showing acute or subacute uveitis, with reduction of tension to normal in only 17. The tension of 2 eyes was not made normal with either atropine or pilocarpine.

Paracentesis was performed on 58 eyes showing some degree of active uveitis. It was surprising to find that normal tension was obtained in only 25 eyes without a more radical surgical procedure. In only 6 of these 25 eyes was paracentesis repeated more than once when it was the only operation necessary. This may suggest that some other procedure might better be utilized if paracentesis fails on two occasions.

In a summary of 60 cases in which iridectomy was done as an initial or as a final measure, it was found that in 43, or 71.6 per cent, the operation was successful and in 17, or 28.3 per cent, it failed. On the whole, gratifying results in cases of glaucoma secondary to primary iridocyclitis are obtained with iridectomy when the procedure is indicated because of failure of medical therapy or the conservative method of paracentesis.

A single trephination was done on 15 eyes. The intraocular tension was reduced to normal in 9 of these eyes, and none showed any activation of the iridocyclitis. Trephination was the initial operation in 8 instances, in 4 of which it was successful. The trephine operation had been preceded by single or multiple operations of other types on 5 of the 9 eyes in which it apparently controlled the tension. In 4 instances trephination was preceded by iridectomy and in 1 by paracentesis.

Miscellaneous operations were performed, usually as a last resort, in a number of cases in which the prognosis was very poor or other operations had failed. We were surprised to find 23 successes and 14 failures in this group. Cyclodialysis was done on 18 eyes in a quiescent state, and in 10 eyes the tension was reduced to normal during the period of observation. Two transfixions of the iris for *iris bombé* were successful, and 1 failed. Twelve combined extractions for cataract complicated by iritic glaucoma resulted in normal tension in 9 eyes. Iridenclesis as the primary procedure was successful in 2 eyes and failed in 2 eyes after trephination.

Sixty-seven eyes of this series were enucleated. The condition of 35 was hopeless, and enucleation was the primary procedure. Eight other eyes were considered hopeless, but at the request of the patients, medical therapy was tried, without success, for a short period before enucleation. On the remaining 24 eyes one or more surgical procedures were carried out before the condition was given up as hopeless.

DISCUSSION

Dr. Peter Kronfeld, Chicago: One of the many annoying and exasperating features of iridocyclitic secondary glaucoma is the extreme variability and irregularity of the disease. If in a case of iridocyclitis the intraocular pressure becomes elevated, it is difficult to predict whether this condition will be just a brief episode

or whether it will prove to be permanent secondary glaucoma, resistant to all forms of medical and surgical therapy at one's disposal. Even the gonioscopic findings do not always enable one to tell with certainty whether the anatomic alterations in the angle of the anterior chambers are such that the function of the normal channels of outflow is permanently or irreparably impaired. Thus, in cases of iridocyclitic secondary glaucoma one has to act from day to day, or rather from week to week, since, with the one exception of seclusion of the pupils with *iris bombé,* no situation associated with iridocyclitic glaucoma calls for immediate surgical action. The well known tolerance of the human eye to such elevations of intraocular pressure and the possibility that nature will come to one's help justify the conservative attitude which is so well expressed by the term "sitting tight."

I have seen several cases of more or less quiescent iridocyclitis in which an elevation of intraocular pressure, despite intensive medical treatment, persisted for as long as eight months and then subsided, with hardly any permanent loss of visual function. The experiences of my associates and myself at the Illinois Eye and Ear Infirmary are in agreement with those of Dr. Fralick. In treatment of glaucoma associated with quiescent iridocyclitis, cyclodialysis has in our hands scored more successes than a trephine operation. The difference, however, is not great enough to permit the statement that cyclodialysis is the most suitable operation for this type of glaucoma, nor do we have any data, gonioscopic or clinical, which enable us to tell beforehand which of the two operations, trephination or cyclodialysis, is more likely to be successful in a given case.

In a case of extensive peripheral anterior synechia, especially in the upper portion of the angle, I prefer cyclodialysis to trephination.

In each of Dr. Fralick's series of 5 cases cyclodialysis after iridectomy was successful. I wonder whether he attaches any significance to this observation. Was the cyclodialysis made in the area of the coloboma? If so, the success of these operations may have been due to the effect of the iridectomy combined with the cyclodialysis, the procedure advocated by Czermak, Wootton and Wheeler, and recently by Goldmann and Bangerter. In Dr. Fralick's series, iridectomy alone made an unusually good showing. I wonder if extensive posterior synechiae were characteristic of the cases in which iridectomy alone controlled the glaucoma.

I should like to take issue with one point in this paper, namely, the use of the word "only" in the following sentence: "Paracentesis was performed on 58 eyes showing some degree of active uveitis. I was surprised to find that in only 25 eyes was normal tension obtained without a more radical operation" Does such a percentage of successes in a procedure as simple and harmless as paracentesis justify the use of the word "only," when a much more extensive operation, such as trephination, was successful in 60 per cent?

Dr. Fralick's experience that paracentesis repeated several times at short intervals does not tend to be any more effective than one or two paracenteses agrees entirely with ours. It is possible, therefore, that paracentesis acts largely by breaking a vicious circle. Our experience with the cyclodiathermy of Vogt is sufficiently encouraging to warrant recommendation of its trial when other surgical procedures have failed.

The period of postoperative observation in our cases has not been long enough to warrant my presenting any results.

In my opinion, Dr. Fralick and his associates have made a contribution of great practical value in a field where every bit of guidance and advice is keenly appreciated.

Dr. F. Bruce Fralick, Ann Arbor, Mich.: Dr. Kronfeld asked whether the cyclodialysis opening was made in the region of the coloboma of the previous iridectomy. I cannot answer that question definitely, but it is my impression that the opening was not uniformly made in the region of the coloboma but was more commonly placed in the outer lower quadrant.

Dr. Kronfeld also raises the question whether posterior synechiae were not common in the cases in which iridectomy was performed. It is true that the patients requiring iridectomy, or on whom irridectomy was done, did show a high percentage of relatively advanced posterior synechiae, and that fact may logically be an explanation for the high percentage of good results in that group. However, this group did not show the characteristics of *iris bombé*, so that the end result would be that of a transfixion.

We also have been favorably impressed by the results of cyclodiathermy cauterization in treatment of the quiescent form of iridocyclitic secondary glaucoma. I have, however, not used it on eyes with good vision. Most of the patients operated on by this procedure have been persons who did not respond to the other forms of treatment, and thus cyclodiathermy cautery was selected as a last resort. We have been favorably impressed with the results in this particular group and may later gain sufficient courage to extend the use of the operation to the patients with less hopeless conditions.

Postoperative Exotropia with Limitation of Adduction: Results of Transplantation of Tenon's Capsule. Dr. Conrad Berens and Dr. Hunter H. Romaine (by invitation), New York.

The technic of obtaining a suitable Tenon capsule graft to be placed between the muscle and the scleral adhesions, although described in a previous publication (*Am. J. Ophth.* **21:** 536, 1938), will be discussed in the light of added operative experience.

In the case of a patient operated on by one of us (C. B.) the first operation was a failure, and the operation had to be repeated for postoperative exotropia following retroplacement. This was done, with an excellent result, after consultation with Dr. Kirby and Dr. White. In another case the exotropia was worse after transplantation of the capsule; the unfavorable result seemed to be due to a reaction to buried catgut. The results in the other cases in which one of us (C. B.) operated were fair to good. In the majority of cases one or more operations had been performed to correct the secondary deviation before Tenon's capsule was transplanted.

In cases of postoperative exotropia, after the muscle is freed from underlying adhesions, it has usually been found advisable to resect a piece of the muscle and advance it about 1 mm. anterior to the original insertion. The results are not ideal, and better methods of handling the Tenon capsule graft to prevent reformation of adhesions between the muscle and the sclera may be devised, or other material, which will increase the effectiveness of the operation, may be found which will prove more satisfactory.

We have performed experimental studies on rabbits directed toward improving technic, clarifying clinical findings and possibly obtaining a better substance for the graft.

Patients with postoperative exotropia (caused by adhesions) are so disfigured that any operation which offers hope for improvement seems worthy of trial, especially in view of the generally unsatisfactory results of conventional operations.

DISCUSSION

Dr. A. D. Prangen, Rochester, Minn.: This paper by Dr. Berens and Dr. Romaine is a valuable contribution to surgery of the extraocular muscles. First, it offers a new and feasible method of dealing with postoperative adhesions which interfere with mobility. Second, and of most interest to me, it points to the significance of Tenon's capsule and the value of preservation of its function when operations are performed on the extraocular muscles.

I agree with the conclusions of the authors that the sliding flap, or, better, the apron flap, is the best procedure. By this method less tissue is damaged; fewer sutures are used, and the exposed muscle is enclosed by its original loose

fascial envelope, through which it can slide. The results of this operation, as reported in this paper, are not striking but are encouraging. When cases of limited postoperative mobility due to adhesions are encountered, this procedure should be tried.

In cases of limited postoperative mobility from adhesions, one wonders why such complications occur. I believe they are caused either by unnecessary damage to Tenon's capsule at the time of operation or by the surgeon's overdoing the original operation. The latter cause is obvious and, I assume, does not explain the postoperative cicatricial loss of mobility considered in this paper. The first cause, disturbed function of Tenon's capsule, is the point under discussion. Throughout the years I have watched operations on the muscles, my own work and that of others, and I gradually have come to feel that coarse carpentry will not suffice for the delicate mechanism of the extraocular muscles. The regional anatomy and physiology of the several tissues involved in the function of the extraocular muscles must be understood and respected. Of these tissues, the fascia bulbi, or Tenon's capsule, is of paramount importance. My consciousness of the importance of this capsule was awakened by the late Dr. P. C. Jameson.

The studies on the anatomy and physiology of Tenon's capsule of rabbit eyes made by Dr. Berens and his associates are most informative. They remind one that Tenon's capsule is a sheet of loose fibrous connective tissue enveloping the muscles, a synovial membrane through which the muscles slide. The authors state: "It is significant to note that by even slight manipulation of the muscle, such as would be made in the usual resection or recession, numerous adhesions were produced without the aid of other implements." Postoperatively, every rabbit's eye showed adhesions of the muscle to the sclera, but these adhesions were less between Tenon's capsule and the sclera than between other tissues and the sclera. Moreover, even when the capsule was adherent to the conjunctiva and the sclera, the loose structure of the capsule allowed the muscle to slip through it.

It is clear, therefore, that in operating on the extraocular muscles the surgeon should seek to effect minimal trauma to the portion of Tenon's capsule on the muscle proper and should try to keep the fascial envelope of the muscle intact during resection or recession. With care, this is possible. When the conjunctiva is opened, Tenon's capsule on the muscle can be avoided. When the tendon and muscle are isolated, sharp cutting dissection parallel to the muscle, instead of tearing blunt dissection, can be used. The latter method tends to pull the capsule off the tendon and muscle, particularly if the capsule has been opened by the incision. The tongue of tendon and muscle with the intact capsule then can be altered *en masse*. This, as Dr. Berens and Dr. Romaine have shown, invites fewer scleral adhesions, and, even though adhesions occur, the muscle will slide through its original capsule. I know this to be true from my own experience. It is the reason for my rejection of any operation which advocates destruction of Tenon's capsule in an attempt to improve or preserve mobility. As a further means of lessening the trauma of tissue and adhesions, I have come to use as few tendon sutures as possible and to employ fine no. 000 and no. 0000 plain gut sutures, which cause less reaction but hold long enough for union to occur. Also, I avoid burying these sutures by tying the knots outside the conjunctiva.

For cicatricial loss of mobility I believe the operation, as advocated by Dr. Berens and his associates, is feasible and, as I said previously, should be tried. No doubt, for the reasons already stated, Tenon's capsule is the tissue of choice for correction of these adhesions when a transplant is needed.

DR. CONRAD BERENS, New York: First, permit me to thank Dr. Prangen for his excellent discussion and for his stressing of the importance of preservation of Tenon's capsule in the ordinary, everyday operations on the muscles that the surgeon has to perform. His method of operation is one which I have tried to follow for some time, but I confess that since postoperative cicatricial strabismus occurred in 1 case of mine, I have been doubly careful.

Dr. Prangen asked whether we were describing the ordinary overcorrection. I have had, and have reported, many overcorrections but not, I am glad to say, this type of deviation, in which we found the extensive adhesions between the muscle and the eyeball, with almost complete limitation of movement in the field of action of the affected muscle. One case of cicatricial strabismus is a low incidence in view of the fact that in the early days I was taught to cut into the capsule and to snip along the sclera and that I did it rather freely. In the light of the experiments on animals that my colleagues and I have performed, I do not see why I have not had a great many more of these unfortunate complications.

Results of Irradiation of Malignant Melanoma of the Uveal Tract. Dr. Frank E. Burch, St. Paul, and Dr. Walter E. Camp, Minneapolis.

The statement has often been made that irradiation of malignant melanomas of the uveal tract is useless. No conclusive proof has accumulated to commend or to condemn this form of therapy as a means of "sterilization" or of destruction of the neoplasm, preliminary to enucleation. The employment of radium after enucleation is frequent when it is available, the idea being that it may prevent local recurrence or metastasis.

In order to afford some evidence of the efficacy of cross fire irradiation of the globe and orbit, as well as the changes in the melanoma produced thereby, sections of irradiated ocular melanomas were studied and compared with similar tumors not so treated. This study seemed worth while, in view of the impossibility of one's determining the degree of malignancy by biopsy, by cytologic study or by the presence and degree of argyrophilic fibers or, in most instances, by predetermination of the probable age of the tumor at the time of the first diagnosis.

No attempts were made to destroy the tumor by application of radium, although several patients in this series refused enucleation until glaucoma, orbital extension or metastases developed. With 1 patient whose affected eye was the only useful one, cross fire irradiation and application of radon were employed, and for a time growth was retarded. Glaucoma subsequently necessitated enucleation.

Our purpose in employing cross fire irradiation under highly competent direction was to determine the possibility of one's safeguarding the patient against local or systemic recurrence.

The results prove that malignant melanoma is not arrested by therapy preliminary to enucleation; that malignant melanoma of the uveal tract is radioresistant to a dose which may be safely employed, and moreover, that although follow-up evidence as to rapidity of metastatic recurrence in the liver, lungs or other organs is difficult to obtain, the mortality rate may be increased by preliminary irradiation, a possibility which is to be considered.

DISCUSSION

Dr. A. B. Reese, New York: The authors speak of the poorer prognosis for malignant melanoma elsewhere in the body than for that in the eye. They cite Bloodgood's figures of only 1.5 per cent of cures in 200 cases of malignant melanoma in other parts of the body, as compared with the approximately 50 per cent of cures in cases of intraocular malignant melanoma. This discrepancy is due not to the difference in location of the same tumor but to the fact that two different tumors are called by the same name. Malignant melanoma in other organs of the body differs from intraocular melanoma not only in regard to prognosis but in regard to its clinical and histologic characteristics. The one seems to be neurogenic and the other ectodermal in origin.

It seems to me the only factor common to these two tumors is the occurrence of pigment. In 39 of the authors' cases glaucoma was present, and in 12 of these the diagnosis of malignant melanoma was not made until the globes were opened in the laboratory, 2 being phthisical. This is not an uncommon experience. It

emphasizes, therefore, the possibility that an eye with long-standing glaucoma may harbor a malignant melanoma, and it serves as another reason for the enucleation of such eyes.

Sometimes statements are perpetuated in the literature without any proved scientific basis or without sufficient data. Such has been the case with regard to the effects of irradiation on intraocular malignant melanoma. Dr. Burch and Dr. Camp now give accurate information indicating that irradiation of such a tumor is without benefit in arresting the growth, preventing metastasis or prolonging life. They admit that a longer period of observation would be advisable. Although their observations are negative, it is important to have them established in the literature on a sound basis.

Angiomatosis Retinae: Report of Successful Treatment of One Case. Dr. Philip Meriwether Lewis, Memphis, Tenn.

This paper was published in the August 1943 issue of the Archives, page 250.

DISCUSSION

Dr. Lawrence T. Post, St. Louis: Dr. Lewis is to be congratulated on the successful treatment of the local ocular manifestation of this rare disease. Though the destruction of one lesion in a case of this disease, in which there may be multiple lesions, may only postpone the evil day, as the author has pointed out, it is to be hailed as a great achievement and might, under more favorable conditions, prolong useful vision for many years.

The desirability of the addition to the literature of new cases of this rare disease is a point well taken. It requires a long time for the clinical picture of an unusual condition to become so familiar to all members of the medical profession that they will be on the alert to recognize the signs when encountered. Probably this disease is not so rare as has been thought.

I am reporting 3 cases here, 2 of which I am sure have not been described in the literature, and Dr. R. H. Boots, former resident at Washington University School of Medicine, has encountered 3 other cases in the examination of draftees at Jefferson Barracks in the first six months of his service. I shall now show photographs of 1 of Dr. Boot's patients. It is obvious that when a case of a disease has once been seen and recognized, new cases of a similar nature are much more likely to be identified.

The first case that I shall describe is that of E. C. R. a married woman aged 30, of Dallas, Texas, who was referred by Dr. J. Guy Jones on April 19, 1937. She gave a history of dimness of vision in the right eye of four months' duration. Vision was 20/24 in the right eye and 20/15 in the left eye.

Just temporal to the right macula there was an area of healed inflammation or hemorrhage, measuring 1 disk diameter horizontally by 0.5 disk diameter vertically, over which the retina was somewhat elevated. The descending temporal vein was dilated to about twice its normal size. The accompanying artery was beaded for a distance of 4 disk diameters below the disk and then showed aneurysmal dilatation to about twice its previous size. It could not be differentiated from the vein. The artery and the vein terminated in a silvery gray mass, which was elevated 5 or 6 D. At the apex of the mass was an orange ball, into which the artery and vein led directly. The top of the mass measured 6 D. The disk was somewhat blurred and showed hypergliosis. The cup was filled with new connective tissue, and all veins were dilated. There were a few hyaloid areas in the lower portion of the periphery. Examination of the left eye revealed a normal macula.

Complete examination was not possible, as the patient was making hurried rounds of the country to find some one who could help her. The correct diagnosis of the condition had been made by the referring ophthalmologist. The patient brought excellent illustrations of her fundi, both of which were extensively involved with characteristic vascular lesions and multiple "bee's nest" tumors, and the

retina of the right eye was widely detached. I advised her that radium or high voltage roentgen radiation offered the best means of therapy that I knew. Her next stop was to be in Kansas City, where she was to see Dr. Curran. Whether her case has been recorded previously I do not know. An interesting feature of the case was that a brother, whom I did not see, was similarly affected. His case was described in a letter to me by Dr. J. Guy Jones. Their father had one blind eye with a white pupil. I wrote her a month ago to find out what had happened since I saw her, but the letter was returned unclaimed.

My second case was that of E. D., referred by Dr. Boots from the St. Louis induction center (slide). The greatly enlarged artery and its accompanying giant vein can be well seen. In this case there were no tumors visible and no evidence of cerebellar or other involvement; hence the diagnosis is presumptive. No treatment was given.

The third case was that of B. K., a single woman aged 27, whom I first saw on May 16, 1942. She complained of decreasing vision in her better eye over a period of two weeks. The other eye had always been defective and on examination proved to be moderately amblyopic, with vision of 20/50 and relative central scotoma. This fact, naturally, was an ever present threat to any contemplated therapy.

Vision of the left eye, which contained the angiomatous tumor, was 20/30 (slide). Obviously, the lesion was all in the extreme posterior pole of the eye near the disk and macula, which fact also influenced therapeutic considerations. Edema and exudate had already extended into the macula, involvement of which caused the blurred vision which the patient complained of.

Three methods of treatment seemed worthy of consideration: application of radium seeds, diathermy puncture and application of thermophore. The preference was in the order named, chiefly because of the inaccessibility of the tumor. An earnest wish not to damage the macula further and not to rupture a large vessel in this, the patient's only eye, was never out of the surgeon's mind.

There was only one week in which the patient was available for treatment, because of her personal affairs. Radium seeds were ordered from New York. As ill luck would have it, they did not arrive as planned. Preparations were made, therefore, to use diathermy, the convenient long curved Gradle needle, devised for detachment of the retina, being chosen. Again, bad luck intervened, and this needle could not be found. Hence, the thermophore was finally used. The awkwardness of the approach through the necessarily inadequate exposure was the real deterrent to the operation. The slide shows the tip employed. I devised this instrument some years ago for treatment of retinal detachments. The elbow at the turn near the point and the size of the shaft, necessary to convey adequate heat to the tip, prevented ready accessibility to the sclera opposite the growth. The eye had to be rotated downward, so that direct vision during application was not possible. Neither could the effect be observed immediately because heating at 160 F., a temperature which will destroy choroid and which it was thought would destroy the tumor, does not produce any immediately observable effect. Therefore, if the application had not been made at the desired point, another trial could not be made immediately. After a prolonged struggle, the point was placed over what was thought to be the correct area, though the attempted scarification of the localized spot had not been satisfactory. The optic nerve could easily be felt, and it was known that the tumor was approximately 4 mm. almost directly above it. What was not realized was the possible rotation of the ball in fixation of the globe. I was chagrined to find on examination of the fundus a few days later, when the great reaction had subsided, that the round white area corresponding to the surface of contact with the thermophore was clearly visible just below and temporal to the macula! No opportunity for a picture to show this offered itself, and when the fundus was next seen, this white area had entirely disappeared and the condition of the eye seemed the same as before the operation.

Encouraged by Dr. Lewis' success, I may try again, using diathermy, though I hesitate to risk further damage to the macula, which is so near the tumor.

DR. JACK S. GUYTON, Baltimore: I wish briefly to present 2 cases in support of Dr. Lewis' conclusions.

The first case is that of a 13 year old girl with bilateral angiomatosis retinae. The patient has been followed by Dr. F. H. McGovern, of Danville, Va., during the past year. Under his direction her eyes were first treated by roentgen irradiation. This failed to check progression of the lesions, and during June 1942 he referred her for diathermy operations.

I shall confine myself to the results of operation on the right eye. The corrected vision in the eye prior to operation was 20/15 — 2. The single angioma was localized by flashing the light of an ophthalmoscope on the lesion and marking the point where the light penetrated the sclera. Sixteen Walker pins were inserted, as in a detachment operation, into and just around the tumor mass, their position being confirmed by frequent ophthalmoscopic examinations. A current of approximately 15 milliamperes for three seconds was used for each pin. A small hemorrhage appeared within the tumor during this procedure. No attempt was made to close off the nutrient vessels, and at the close of operation the vessels appeared almost as large as before, although the angiomatous mass had been completely obliterated.

Postoperatively, the fundus showed a pronounced reaction. A moderate-sized hemorrhage surrounded the obliterated angioma, and in the overlying vitreous a good deal of subretinal serous exudate was present, producing a flat detachment of the entire upper half of the retina and some subretinal hemorrhage around the temporal side of the disk. This reaction slowly, but completely, subsided. Three weeks after operation the corrected vision was 20/50 — 1. The retinal vessels were gradually reduced to normal size within the first week after operation. Six weeks after operation all hemorrhage and exudate had disappeared; the site of the angioma was a healed scar, and corrected vision had returned to 20/15 — 2.

The second case is that of a 7 year old girl with bilateral juvenile Coats's disease. Progressive failure of vision developed in her left eye during the summer of 1941. She was first examined in September of that year, at which time the left eye was completely blind, with advanced exudative retinitis. This eye was eventually enucleated, after a short episode of secondary glaucoma. Histologic examination showed in the periphery of the retina on one side some unusually large blood vessels associated with an area of massive glial proliferation, and in places a large number of capillaries. There was massive subretinal exudation. There were newly formed vessels on the anterior surface of the iris and ectropion uveae. Although the histologic picture was that of massive exudative retinitis and glial proliferation (Coats's disease), it suggested atypical angiomatosis retinae.

In September 1941 vision in the right eye was 20/20, and the eye was entirely normal except for the fundus. The disk was slightly grayish and definitely blurred. In the far temporal periphery was an area of grayish white exudation, about 2 disk diameters in extent. The vessels over this area were small but showed sharp and irregular changes in direction, as if affected by local elevation of the retina.

During the succeeding months the exudation in the periphery of the right eye gradually increased; also, definite beading of vessels became evident in that region, and a small amount of hemorrhage appeared within the exudate.

During March 1942 the right eye was given 930 r of roentgen radiation in divided doses; a current of 200 kilovolts was employed, with a 2 mm. copper and a 1 mm. aluminum filter, and with a small aperture to avoid irradiation of the lens. In spite of this therapy, the area of exudation gradually, but steadily, increased; the disk became much more edematous, and a hazy spot of subretinal exudation began to form just below the disk.

The right eye was operated on Aug. 1, 1942. The area of exudation in the temporal portion of the periphery was completely obliterated and surrounded with sixteen diathermy punctures, Walker pins being used as in a standard detachment operation. During the first week after operation there was surprisingly little reaction in the fundus. Eleven days after operation corrected vision was still

20/20 — 3. An elevated white area, containing some blood undergoing absorption, extended from 7 to 10 o'clock in the temporal periphery. The disk was slightly more edematous and the subretinal exudate below the disk was slightly more prominent than before operation. Four weeks after operation the reaction in the fundus reached its height, with increased edema of the disk, which extended into the macular region. At this time vision with a pinhole disk was reduced to 20/70. During the succeeding four weeks the area in the periphery that had been treated with diathermy punctures contracted into a white scar with a pigmented border. The edema of the macula and about the disk disappeared; the subretinal exudation just below the disk was absorbed, and corrected vision returned to 20/20 — 3.

There has heretofore been no treatment of any kind for juvenile Coats's disease. The apparent success of diathermy punctures in this case is therefore of considerable importance. There has been a good deal of argument as to whether the pathogenesis of juvenile Coats's disease is the same as, or similar to, that of angiomatosis retinae, but whatever the primary cause of the lesion may be, the observations here reported indicate that obliteration with diathermy at least offers a chance of the vision's being saved. It is significant that in this patient eradication of the primary lesion in the periphery of the retina caused disappearance of the edema of the disk and of the subretinal exudation below the disk.

DR. FREDERICK C. CORDES, San Francisco: It is only recently that the treatment of angiomatosis retinae has received much attention. In addition to the method employed by Dr. Lewis, radium and roentgen rays have been used. Treacher Collins, in 1930, proposed electrolysis as a possible means of therapy and suggested that localization of the tumor be made according to the methods employed by Gonan. Roentgen radiation has been employed a number of times.

There are few reports of cases in which the patient was observed more than a year. Weve reported his results two and a half years after diathermic treatment, and Craig, Wagener and Kernohan published their observations three years after roentgen therapy.

In 1940 Dr. M. J. Hogan and I (Angiomatosis Retinae, ARCH. OPHTH. **23:** 253 [Feb.] 1940) reported our results in a case of fairly early angiomatosis retinae in a woman of 20, who received 1,202 r of roentgen radiation in September 1938. In the left eye a lobulated, reddish yellow mass was situated well out in the periphery below and slightly to the temporal side. The inferior temporal artery a short distance from the disk was greatly dilated and tortuous. In the central portion the artery became sacculated. Accompanying the artery was a tortuous, but more dilated, vein, which started in the mass. There were patches of pale yellow exudate above the macula and a good deal of edema and many linear streaks of exudate along the vessels.

The patient was observed periodically until she moved to Seattle. She retained the improvement described in the paper. In Seattle the patient has been under the observation of Dr. Will Otto Bell, who has kept us informed on the results of her periodic examinations. His letter written three and a half years after the irradiation was started states:

"The sacculation and tortuosity mentioned in the report have notably receded. The vein remains somwhat enlarged. The mass measures almost 2 disk diameters and is elevated from 3 to 4 D. No other masses have been seen. The patches of pale yellow exudate above the macula mentioned in the original report could not be found, and the linear streaks of exudate above the dilated vein are absent." Vision remains the same as before treatment—0.8 + with correction. Interestingly, this reduction in vision is due to an opacity of the lens that she had before irradiation and which has not increased as a result of the treatment.

My associates and I also have under observation a 14 year old girl with bilateral angiomatosis retinae. The condition is well advanced in the right eye and is in the early stage in the left eye. Her father died at the age of 35, of Lindau's

disease. Aside from the lesions in the eyes, the patient shows no signs of the disease. In February 1941 she received 1,800 r of roentgen radiations to each eye, a dose twice that which Dr. Guyton gave his patient. She was last seen in September 1942, two weeks ago, and nineteen months after irradiation was started. The condition in the right eye seems to have definitely progressed. In the left eye the nodule is smaller, white and "harder," as though it consisted for the most part of scar tissue. Vision in the left eye remains at 20/20.

Cases of well advanced disease have been reported in which roentgen therapy was not successful. In our experience this observation seems to be borne out. In the second case the well advanced stage in the right eye showed no regression. The early stage of the disease in case 1 and that in the other eye in case 2 did respond to roentgen therapy, and the eyes have maintained their improvement and function, one for three and a half years and the other for one and a half years.

There still persists a widespread fear of roentgen therapy in treatment of ocular conditions. The most dreaded complication is lenticular opacities, a condition that can be corrected by operation and therefore should not be considered too seriously. Clapp could find only 34 cases of postirradiation cataracts up to and including 1932, a period covering the time when irradiation of an eye was not as well understood as it is now.

On the basis of observation of 3 eyes, 1 of which was irradiated and observed for three and a half years and the other 2 for one and a half years, we feel that in addition to diathermy, roentgen irradiation offers a convenient, safe means of treatment of the lesions of this disease if they are seen early, before gliosis has developed.

DR. F. BRUCE FRALICK, Ann Arbor, Mich: I am pleased that Dr. Lewis has again called attention to the fact that angiomatosis retinae in some instances is amenable to treatment. He is to be complimented on his careful observations and his results in this case.

It is my belief that the number of patients operated on for this condition will be small and that therefore every surgeon should place on record his results in each operation. I wish, therefore, to report an additional case of angiomatosis retinae.

A 26 year old man was referred to me by Dr. Warren B. Dodge Jr., of Battle Creek, Mich. The patient had experienced no visual complaint until eight months before, when he noted blurred central vision in the right eye. There were no subsequent visual changes and no inflammation. The general health was good, and the family history was without significance. General medical and neurologic examinations revealed a normal status. Vision was 6/30 in the right eye and 6/45 — in the left eye. External and fundic examination of the left eye revealed nothing abnormal. Examination of the right eye revealed normal pressure and no external evidence of disease. Inspection of the fundus of the right eye revealed that the superior temporal artery and vein were much dilated and tortuous. The vessels terminated in an angiomatous cyst in the upper outer periphery of the retina. The superior temporal quadrant of the retina was studded with small lipid flecks. The macular area was infiltrated and edematous and contained many lipid infiltrates. The central field of vision presented a scotoma in the lower mesial quadrant, which included the area of fixation.

On April 29, 1941 a diathermy operation for angiomatosis retinae was done according to the method outlined by Herbert Kaye (Treatment of Angiomatosis Retinae, ARCH. OPHTH. **25:** 443 [March] 1941). An attempt was made to obliterate the angiomatous retinal cyst with micropuncture pins and to sclerose the superior temporal artery, which supplied the cyst, by diathermic treatment of the scleral surface along the course of the artery. The latter procedure was difficult, since the artery and vein were relatively close together. An attempt was made to produce the fundic picture of arterial, rather than of venous, occlusion.

When the patient was last seen, on Feb. 17, 1942, ten months after the operation, vision had improved from 6/30 to 6/15 + in the right eye. The dilated vessels were much smaller than before operation, but the superior temporal artery and vein were still patent. The angiomatous cyst appeared as a productive exudative scar, supplied and drained by the superior temporal vessels. The lipid exudates had nearly all disappeared. The retina was flat, and the hemorrhages had been absorbed, without evidence of their former presence.

This limited experience with a condition for which formerly no therapeutic measure was available offers hope for further improvement in surgical technic and results.

DR. ARTHUR J. BEDELL, Albany, N. Y.: I wish to demonstrate by means of colored photographs of the fundus that angiomatosis passes through certain stages whether it is treated or not and, further, that these stages do not fall into definite intervals of time.

(Slide) The first photograph shows an unusually large vein in the inferior portion of the fundus. I believe it is safe to say that when there are very large, parallel blood vessels, angiomatosis is present.

(Slide) The second photograph of the same eye shows the macular exudate and the greater dilatation of both the vein and the accompanying artery.

(Slide) The third photograph, that of the extreme temporal periphery, shows a typical angioma; a rounded, pale, red swelling, with a paler area below and a pinker, upper outer edge, with entering artery and draining vein.

The condition in the second case was in a more advanced stage. The angioma and the blood vessels were small, but the exudate covered a greater area and in the lower part of the fundus the retina was detached. In the case which Dr. Lewis presented, retinitis proliferans followed treatment. I am sure that when he reports on the case in another year or two, he will say that the folding and proliferation of the retina have increased.

(Slide) The photograph in the third case exhibits typical changes, with much pigmentation and several retinal ridges.

Angiomatosis is much more common than the reports indicate. I have seen 20 cases and feel certain that others may have been unrecognized.

Why Is a Glaucoma Clinic Desirable? DR. HARRY S. GRADLE and DR. H. ISABELLE MCGARRY (by invitation), Chicago.

The glaucoma clinic of the Illinois Eye and Ear Infirmary was established because it was believed that constant supervision of patients suffering from glaucoma would result in a notable decrease in blindness due to that disease. To prove the validity of that belief the following paper presents an analysis of the cases of patients with primary glaucoma who were seen at the clinic during the first year of its existence and who have been under continuous observation ever since. The analysis is based on data on visual acuity, visual fields and tension after six, twelve, eighteen and twenty-four months, as compared with the status of these factors at the time of the patient's admission. The series comprises all cases of primary glaucoma, not merely selected cases, but does not include any case in which the etiologic factors of the hypertension could be proved clinically.

The statistics present evidence that the glaucoma clinic, functioning as a supplement to the regular ophthalmic services of the Illinois Eye and Ear Infirmary, has more than proved its value in the preservation of visual function and in the prevention of blindness in cases of primary glaucoma.

DISCUSSION

DR. MARK J. SCHOENBERG, New York: Last year the chairman of the Committee on Glaucoma for greater New York, sponsored by the National Society for the Prevention of Blindness, read before the American Ophthalmological Society a report on a survey of glaucoma records of three ophthalmic clinics in New

York and of one such clinic at Boston. The greater part of this paper illustrated how unsatisfactory and chaotic glaucoma work can be if it is conducted along traditional lines, which have remained almost unchanged for the last one hundred years. On the other hand, study of the Boston clinic demonstrated that it is not impossible to reform the old ways and that better results are obtainable with more up-to-date methods, just as Dr. Gradle and Dr. McGarry have emphasized in their paper. I wish to add that conditions are improving fast in the ophthalmic clinics of New York.

As an introduction to discussion of the paper, I shall relate some of my own experiences.

In October 1935 my colleagues and I opened a special clinic for patients with glaucoma at the Knapp Memorial Eye Hospital, of New York. The staff of the clinic consisted of a director, an assistant and a medical diagnostician. This group started out with the idea of studying the patients as thoroughly as the means at our disposal would permit. Perimetric measurements were recorded every four to six weeks; the patient's complaints, relating both to the physical and to the mental condition, were entered on the record, no matter how trifling they appeared.

In addition, each member of the staff was charged to study and report on a certain phase of glaucoma. Gradually the work branched out in every direction. A social worker and a nurse were added to the staff, and patients were taught how to apply medicaments to their eyes and the elementary rules of personal hygiene and nutrition. The meaning of glaucoma was explained and the importance of cooperation stressed. Printed leaflets containing the necessary information, worded in plain language, were distributed among the patients and the members of their families. The clientele of the clinic grew gradually. Thus by organizing a glaucoma clinic, coping with the various practical problems and handling patients in a manner entirely different from the way in which they are being managed in regular clinics, and even in private practice, we learned some things not found in books or in journals. From this experience we reached the following conclusions:

1. The glaucoma clinic gives an infinitely better service to patients than the average ophthalmic clinic.

2. The clinic offers the attending staff, both medical and nonmedical, the rare opportunity of studying and treating a large number of patients with the same disease within a short time, so that the span of time necessary to acquire a sufficient amount of knowledge about glaucoma is shortened.

3. The clinic serves as a center of education for patients and other lay people who are in need of being informed about the prevention of blindness from glaucoma.

4. The clinic may serve as a center of education for medical practitioners and others, who are the first to come in contact with patients with early glaucoma and on whom we depend for getting such patients.

5. It could become a center for research, especially clinical research, on the various phases of the glaucoma problem.

In other words, such a clinic offers (1) better service to patients; (2) an opportunity to the clinician for intensive study of patients with glaucoma, and (3) educational opportunities to both layman and general practitioner. It may also serve as a center for research.

The objections to a glaucoma clinic may be briefly considered. Few ophthalmologists will deny the importance of the organization of special glaucoma clinics. No one could defend successfully the traditional method which has been in vogue for the past century. On the other hand, many ophthalmologists doubt whether the organization of special glaucoma clinics is a practical possibility. Some of their objections refer to: (1) the cost of running such a clinic; (2) the lack of medical and nonmedical help; (3) the lack of space, and (4) the opposition of the medical staff of the general ophthalmologic clinic, which does not wish to be deprived of

the experience of seeing patients of this type while a small group of ophthalmologists of the glaucoma clinic "monopolizes" the entire clinical and surgical material of the disease.

Those who have condemned innovations in medicine have always been in the majority, and apparently valid objections have always been found even against the most pressing reforms.

The shortage of well trained professional perimetrists is indeed a serious problem, but even this can be remedied. Lately, my associates and I have started in New York a course for teaching volunteers the method of perimetry. We hope to have half a dozen perimetrists fairly well trained in about three months. They will then be turned over to a few ophthalmic clinics, where perimetry is done only casually, to work and to continue their training under the supervision of the attending ophthalmologists.

A small room 10 by 10 feet (3 by 3 meters) can be fitted out with the necessary implements to serve as a perimetry room. In view of the benefit to be derived from perimetry, the salary of a professional perimetrist is the best investment an ophthalmic institution can make. If there is money for a laboratory, for roentgenography and for departments of orthoptic training and correction of aniseikonia, there must be money for perimetry.

The accusation that a separate clinic "monopolizes" the glaucoma material is based on misinformation and misunderstanding. The staff of the glaucoma clinic is to be on a rotating basis. The members are appointed for three, six and nine months on a staggered basis, so that there are always ophthalmologists in charge to teach the newcomers from the general ophthalmic clinic the routine of the work and to continue the tradition of thoroughness established by the director.

Time does not permit discussion of the subject in greater detail. Men who have had actual experience with glaucoma clinics should have priority in being heard. The opinions of the colleagues from such institutions as the Illinois Eye and Ear Infirmary, the Massachusetts Eye and Ear Infirmary, the Brooklyn Eye and Ear Hospital and the Long Island College Hospital, as well as my own conclusions, should weigh in the balance against those of men who do not approve of special glaucoma clinics on purely theoretic grounds.

Dr. John Evans wrote to me a few days ago as follows: "I can state that there is absolute agreement at the Long Island College Hospital that the patients are getting far better care in the glaucoma clinic than in the general clinic." Dr. Henry Mitchell Smith wrote: "From my experience as director of the special glaucoma clinic at Long Island College Hospital, I am convinced that a special clinic is the only way to handle patients with glaucoma in a busy ophthalmic clinic."

I do not doubt all agree that the real difficulty in running a successful glaucoma clinic lies in the discovery of cases of the disease in the early stages. To do this, the public, the general practitioners and the optometrists must be trained. Research work must be encouraged. The ophthalmologist should not be satisfied simply to prevent blindness from glaucoma. Even 20/100 vision is too low a goal. He should strive to save for his patients normal, or nearly normal, visual acuity and field of vision.

This discussion should not be concluded without my pointing out one weakness in the otherwise excellent paper of the essayist, namely, the statement that the glaucoma clinic at the Illinois Eye and Ear Infirmary neither treats the patient nor operates. In other words, the clinic concentrates its entire attention on and limits its activity exclusively to, diagnosis. I believe that it is much better, and to the advantage of the patients and of the personnel of the glaucoma clinic, that the complete work (both diagnostic and therapeutic) be carried out at the glaucoma clinic only. Divided interest and responsibility do not lead to complete efficiency. Perhaps the possibility that members of the staff of general ophthalmic clinics may fear they are being deprived of opportunity for glaucoma surgery has dictated the authors' policy of splitting the work between the two services. How-

ever, this fear is due only to a misunderstanding, since, as I have mentioned, one of the purposes of the glaucoma clinic is to give an opportunity to every man from the general ophthalmic clinic to become proficient in every phase of the glaucoma problem by devoting his time at least for one period, of a number of months, exclusively to the work in the glaucoma clinic. This includes, of course, glaucoma surgery.

Differential Diagnosis of Paresis of the Oblique and of the Superior and Inferior Rectus Muscles. Dr. William Thornwall Davis, Washington. D. C.

Bielschowsky stated that "by far the most frequent type of single vertical motor anomaly is trochlear palsy."

The most striking sign of palsy of the oblique extraocular muscles, both superior and inferior, is habitual torticollis. The head is not tilted with palsy of the vertically acting rectus muscles. Until Bielschowsky formulated the diagnostic signs for differentiation of palsy of the superior and inferior rectus muscles and palsy of the oblique muscles, paresis of the vertically acting rectus muscles and that of the oblique muscles frequently were confused.

The outstanding differential diagnostic sign may be stated as follows: With palsy of the right superior oblique or of the left superior rectus muscle, when the patient looks up and to the left, there is a high degree of hyperphoria on the right side, due to overaction of the right inferior oblique muscle. With palsy of the right superior oblique muscle, when the head is tipped to the right shoulder, maximum vertical deviation occurs. When the head is tipped to the left shoulder, there is little or no vertical deviation. With palsy of the right superior rectus muscle, there is no change in the vertical deviation when the head is tipped either to the right or to the left shoulder.

There are other diagnostic signs, which will briefly be discussed, but this phenomenon is the most easily observed and the most important. It requires no paraphernalia for detection.

The same difficulty obtains in differentiation of palsy of the right inferior oblique muscle and palsy of the left inferior rectus muscle. When the eyes are rotated to the left, the right eye turns strongly down and in. This is caused by overaction of the right superior oblique muscle, which is the antagonist of the right inferior oblique muscle.

The simplest and most dependable diagnostic sign is elicited by the subject's tipping the head. When the head is tipped to the right shoulder, the side on which the palsy exists, there will be slight or no vertical deviation of the eyes. When the head is tipped to the left shoulder, the maximum vertical deviation occurs. This is the reverse of that which occurs with palsy of the right superior oblique muscle, with which the maximum vertical deviation occurs when the head is tipped to the side of the lesion. With palsy of the inferior oblique muscle, the maximum deviation occurs when the head is tipped to the side opposite the lesion. If the left inferior rectus muscle is paralyzed, there is no difference in the vertical deviation when the head is tipped to one shoulder or to the other.

DISCUSSION

Dr. Sanford R. Gifford, Chicago: Dr. Davis has so well described and demonstrated the principal points in the differential diagnosis of palsies of the extraocular muscles that there is little left to say. There should be no need to insist here on the value of exact diagnosis in cases of paralysis of the vertical and oblique muscles. One still sees, however, a number of patients whose disorder has not been diagnosed or, if diagnosed, no provision has been made for appropriate remedial measures. This is a pity, since few patients present more satisfactory results of appropriate surgical treatment than those with partial

paralysis of a vertical or an oblique muscle. The proof of the diagnosis is in the surgical result, and few patients are more grateful than those who have received appreciable improvement in their binocular field, with relief of long-standing discomfort. Most ophthalmologists are similar in an intractable tendency to forget rules concerning paralytic squint. To have forgotten some of them has been more profitable than to have learned them, for it is true that the textbook picture of diplopia, as shown in nine neat little squares, is rarely met with in practice. It is seen only in cases of fresh paralysis and is supplanted rapidly by the complex resulting from contracture or spasm of the opposing muscle. Dr. Davis has done well to describe this stage of paralysis of the superior oblique muscle, in which the diplopia is not limited to the lower and inner part of the field, but exists all over the field, and the prominent feature is the upshoot of the paralyzed eye in adduction, due to overaction of the inferior oblique muscle. I should go further and say that this is the usual picture and is common in cases in which the superior oblique muscle has so far recovered as to render difficult the demonstration of any limitation of motion downward and inward.

(Slide) This slide illustrates a case of paralysis of the left superior oblique muscle with typical tilting of the head to the right. The eyes are about level.

(Slide) This slide shows the patient looking up to the right, with overaction of the left inferior oblique muscle.

More practical than the remembering of rules, at least in my experience, has been a somewhat childish method by which the position and functions of the various muscles are visualized while one is looking at the patient. Before the field of diplopia is examined, the deviations in various parts of the field in the cover test are noted, as advocated by Bielschowsky, Duane and White, including observation while the head is tipped. Thus, with the action of each muscle in mind, one identifies the muscle which must be paralyzed or overactive in order that the deviation observed may be produced. The only rules worth one's remembering are that in the cover test the deviation of the nonparalyzed eye is always greater than that of the paralyzed eye and that a decided increase in deviation when the head is forcibly tilted nearly always means paralysis of an oblique muscle. I should add one more, namely, that the deviation is always greater in adduction in the case of paralysis of an oblique muscle and is always greater in abduction in the case of paralysis of a vertical rectus muscle.

Bielschowsky's priceless advice is always remembered, namely, that in cases of vertical deviation one should consider only the primary actions of the muscles, as their secondary actions may be neutralized by preexisting phorias. I dislike to think how many diagnoses I have missed by following the textbook picture, which indicated that paralysis of an oblique muscle should result in homonymous diplopia because of the abducting action of that muscle. Almost as often as not, in cases of acquired paralysis, a preexisting exophoria causes the diplopia to be crossed or crossed in one field and homonymous in another. In cases of congenital paralysis of the trochlear nerve convergence is more likely to develop; this may be pronounced enough to stimulate concomitant convergent squint, with overaction of an inferior oblique muscle. This is especially likely to occur when some degree of suppression has developed, as may happen in cases of congenital paralysis, so that diplopia is not noticed. In a child old enough to cooperate, however, diplopia can nearly always be elicited during tests, and in smaller children tilting of the head will always suggest congenital paralysis of the trochlear nerve. The case of a patient with paralysis of the left trochlear nerve whom I recently treated is typical (slide). At the age of 14 years she suppressed the image of the left eye so much that she never complained of diplopia. She was more comfortable, however, when she tipped the head to the right shoulder, and diplopia could be elicited easily when attention was directed to it. In this case the upshoot of the left eye on adduction was the prominent feature, but there was hypertropia in the left eye of 15 prisms in the primary position. (In the picture, she is looking down and in, so that she shows limitation of the motion of the superior oblique muscle

also, but a marked upshoot was evident on her looking straight to the right.) The hyperphoria of the left eye has been reduced to 3 prism diopters by excision of part of the left inferior oblique muscle.

I have cared for a few patients with congenital paralysis of the trochlear nerve in whom the convergent deviation had become so marked as to require surgical correction, in addition to what was necessary for the vertical deviation. In such cases myectomy of the inferior oblique muscle is usually performed first and is followed later by whatever procedure on the horizontal rectus muscles is indicated by the degree of squint.

As Dr. Davis has pointed out, the most difficult problem of diagnosis is differentiation of partial paralysis of the superior oblique muscle and that of the superior rectus muscle of the opposite eye. This is especially true when there is alternating fixation or fixation with the paralyzed eye. The relative positions of the double images are the same in the two conditions. Thus, with paralysis of the left trochlear nerve there is hypertropia of the left eye, which increases on the patient's looking to the right, with the upper pole of the left image tipped to the right. With paralysis of the right superior rectus muscle, there is hypotropia on the right side or hypertropia on the left side, which increases on the patient's looking to the right, with the upper pole of the right image tipped to the left, though usually only slightly. If the patient fixes with the right, or paretic, eye, the image of the fixing eye is seen as erect, and that of the left eye is tipped, with its upper pole to the right. Usually the head tilting test, as described, reveals notable increase in deviation when the head is tipped to the left side in cases of paralysis of the left trochlear nerve, while the degree of deviation is unchanged, or is decreased, in cases of paralysis of the right superior rectus muscle.

(Slide) This slide illustrates another case of paralysis of the left superior oblique muscle, with the head tilted to the right in the normal position and no deviation.

(Slide) An attempt is being made to force the patient's head to the left; the left eye deviates upward when the patient looks to the right with the head tilted.

When there is doubt, it is often of value to note the degree of deviation of each eye in the cover test. In a recent case of paralysis of the right superior rectus muscle when the patient fixed with the left eye deviation was slight, while when he fixed with the right eye, as shown in the slide, the left eye showed a marked upshoot under cover. This, of course, established the right eye as the paralyzed one. Fortunately, myectomy of the left inferior oblique, which is indicated in cases of paralysis of the right superior rectus muscle, is of almost equal value in cases of paralysis of the left trochlear nerve, with overaction of the left inferior oblique; so one may be wrong in the diagnosis and still do the patient some good.

When one is examining for diplopia, which is seldom done until a tentative diagnosis by means of the cover test has been made, the value of the horizontal line should be emphasized. The images of the vertical line, usually employed, often overlap in such a way as to be fused by the patient. Images of the horizontal line, when tipped, are fused much less easily, and hence the patient will give much more reliable answers as to the tipping of images. Its only disadvantage is the necessity of the patient's mentally rotating the tipped image 90 degrees in order to visualize the rotation of the eye.

(Slide) This slide shows a picture of Bielschowsky's, illustrating paralysis of the left superior rectus muscle and the corresponding fields. The patient is fixing with the paretic eye, and the marked upshoot of the nonparetic eye during fixation with the paretic eye is shown. The next picture shows him fixing with the nonparalyzed eye with little deviation. The next shows the normal movement of the right eye on his looking up and to the right, and the last, the marked limitation of motion of the right eye on his looking up and to the right.

(Slide) This picture shows the horizontal line as it is used, and the next, the mental rotation of the image, which one must make in order to visualize rotation

with the paralyzed muscle, which accounts for the tipping. The tipping, of course, is always in the direction in which the paralyzed muscle should move the eye, so that when the left image is tipped with the upper pole to the right, as in this case, there must be paralysis of a right rotator muscle—in this case, the left superior oblique muscle.

(Slide) Once the trick of mental rotation of the image is learned, its advantage is obvious. Many persons will give no information relative to tipping with the vertical line, but do so readily with the horizontal line. The red glass is seldom necessary in such tests, and its use serves only to conceal the deviations which are occurring and which one wishes to observe.

A few slides illustrate this test.

(Slide) This picture was taken in a case of paralysis of the left superior oblique muscle before operation, with a crude picture of the field of diplopia (slide), and the next picture shows the binocular field after myectomy of the left inferior oblique.

(Slide) The next slide shows paralysis of the left superior rectus muscle. When the patient is looking up and to the right, one does not see any deviation. When she is looking up and to the left, there is marked limitation of movement of the right eye in the field of the paralyzed left superior rectus muscle.

(Slide) The next slide (from Bielschowsky) shows paralysis of the inferior oblique muscle. The motion of the left eye is normal when the patient looks up and to the left, but there is complete paralysis of the left inferior oblique when he looks up and to the right.

(Slide) The next slide is a case of mine, one portion of which shows paralysis of the left inferior oblique muscle.

Fixational Corneal Light Reflexes as an Aid in Binocular Investigation. MAJOR EMANUEL KRIMSKY, Medical Corps, Army of the United States.

This paper appears on page 505 of this issue.

Correlation of the Phenomena of Crossed and Obliquely Crossed Cylinders with Cylinder Retinoscopy and Clinical Refraction. DR. CHARLES HYMES, Minneapolis.

Crossed and obliquely crossed cylinders may be resolved into spherocylindric equivalents, which may be determined clinically or by means of precise mathematical formulas. The phenomenon of obliquely crossed cylinders is the basis for cylinder retinoscopy, as well as for the technic of determination of the axis of an astigmatic eye by means of the crossed cylinder.

The only other function of the crossed cylinder in the technic of refraction is that of determination of the power of the cylinder, based on a comparison of diffusion images.

There is no physiologic basis for the use of the crossed cylinder in the determination of the power of the spherical addition for presbyopia.

DISCUSSION

DR. W. HOWARD MORRISON, Omaha: This paper contains too much detail in physiologic optics for one to absorb it readily. Most will benefit by reading and analyzing this contribution after it is printed in the Transactions.

In discussing the simple cylinder, Dr. Hymes uses the formula: $F = \frac{\text{No. of Degrees} \times \text{Power of Lens}}{90}$. This formula, according to Cowan (Refraction of the Eye, Philadelphia, Lea & Febiger, 1938, p. 250), is not exactly correct. It is, however, easy to remember and to use. To be exact it should read as follows: $Dm = Dc \sin^2 a$, in which *Dm* indicates power in any chosen meridian, *Dc*, total cylinder power and *a*, angle between the axis meridian and the chosen meridian.

Dr. Hymes states that as a technic cylinder retinoscopy is not generally used. I should say, rather, that cylinder retinoscopy is employed by most refractionists who use the retinoscope expertly.

Some attention must be paid to the sphere when the cross cylinder is used to determine the strength of the cylinder. He who uses the cross cylinder finds that he cannot increase the amount of the cylinder indefinitely without decreasing the spherical strength. Conversely, he cannot greatly reduce the strength of the cylinder without increasing the amount of the sphere. This means that the cross cylinder is not used in determining the sphere alone, as stated by some refractionists, but it implies that each time more or less strength of cylinder is indicated by the cross cylinder a spherocylindric change must be made.

Cowan also stated (page 268) that with a cross cylinder

> We are able to add either positive or negative cylindric power and at the same time reduce the spheric power in the same denomination and in the same proportionate strength. This differs from the use of plane cylinders for the same purpose.

When making such spherocylindric changes, one must understand the means of obtaining a spherical equivalent; that is, the spherical equivalent of a simple cylinder is equal to one-half the power of the cylinder. For example, the spherical equivalent of a + 4.00 D. cylinder is a + 2.00 D. sphere, or that of a — 5.00 D. cylinder is a — 2.50 D. sphere.

The spherical equivalent of a compound is determined by adding one-half the power of the cylinder to the spherical element of the compound. For example, the spherical equivalent of + 2.00 D. sph. ⌒ + 4.00 D. cyl., ax. 90 is a + 4.00 D. sphere; that of — 2.00 D. sph. ⌒ — 4.00 D. cyl., ax. 180, a — 4.00 D. sphere, and that of + 3.75 D. sph. ⌒ — 4.00 D. cyl., ax. 180, a + 1.75 D. sphere.

The refractionist employing the 0.25 D. cross cylinder uses his knowledge of the spherical equivalent in this manner: If in a combination of + 3.00 D. sph. ⌒ + 2.00 D. cyl., ax. 90, the cross cylinder indicates less strength of cylinder, the next step is to reduce the cylinder by 0.25 D., it being borne in mind that in reality the sphere has been increased 0.125 D. If the cross cylinder again demands that the cylinder be decreased by 0.25 D., then not only is the cylinder again reduced, but the sphere also is altered. The new lens combination then is + 3.25 D. sph. ⌒ + 1.50 D. cyl., ax. 90. One half of the amount the cylinder has been reduced has been added to the sphere.

Prangen (Some Problems and Procedures in Refraction, ARCH. OPHTH. **18:** 432 [Sept.] 1937) stated:

> When it is used for strength the cross-cylinder alters both the sphere and the cylinder, and this alteration should be visualized. If the cylindric effect alone is thought of and the cylinder is changed without the sphere being checked, an increasingly distorted image will be produced, and the interval of Sturm will be exaggerated instead of corrected.

Prangen credited J. C. Copeland, of the Riggs Optical Company, with calling to his attention the rule of spherical and spherocylindric equivalents.

I have had no experience with obtaining by means of cross cylinders the spherical addition necessary for correction of presbyopia. I do, however, understand that some men, particularly optometrists, use a grid chart of horizontal and vertical lines placed at the reading distance and by means of a pair of cross cylinders balance the horizontal and vertical lines with respect to blackness by adding or decreasing spheres in the trial frame. A cross cylinder is placed before each eye with the minus axis at 90 degrees; if the patient with presbyopia needs more correction, the vertical lines will be blacker. Less sphere is indicated if the horizontal lines appear blacker.

DR. CHARLES HYMES, Minneapolis: Several points in Dr. Morrison's discussion of my paper need to be cleared up. He believes that cylinder retinoscopy is employed by most refractionists who use the retinoscope expertly. This does not indicate whether or not that group comprises a majority of refractionists.

Such a recent text as Maxwell's (Maxwell, J. T.: Outline of Ocular Refraction, Omaha, Medical Publishing Company, 1937) states that in cylinder retinoscopy the placing of the correcting cylinder in the wrong axis creates the reflex of mixed astigmatism, which is practically impossible to correct. Maxwell thus inferentially advised against the use of cylinder retinoscopy as a technic. In the body of my paper this very point is discussed both as to its neutralization by means of the correcting cylinder's being shifted toward the true axis of the astigmatism of the eye and as to its actual measurement by means of minus sphere and plus cylinder.

Dr. Morrison apparently confused the clinical spherocylindric equivalents (plural) discussed by Prangen with the geometric spherocylindric equivalent of obliquely crossed cylinders, which is the basis of my paper. Prangen's spherocylindric equivalents are equivalents of a sphere and cylinder and not of obliquely crossed cylinders. In the example cited by him in his fine article and cited by Dr. Morrison, he gave ten different formulas for the writing of what he referred to as an "equivalent prescription." After stating that the full correction of the astigmatism would be ideal, Prangen stated that it is often necessary to give the eye a compromise image or partial correction for the astigmatism by partial obliteration of the interval of Sturm. It is these compromise corrections which he referred to as spherocylindric equivalents, which should be tried before eyes which reject full correction because previously glasses had not been worn. Any one of several of these compromise formulas may be equally acceptable to the same eye.

Perhaps the similarity in terminology was the confusing element, but it is well to emphasize that there can be but one answer to a geometric problem, not several.

I have but one more suggestion to make regarding the use of the cross cylinder in determination of the correctness of the sphere or the spherical addition for presbyopia; that is, cross cylinders refract rays as cylinders, and not as sphere and cylinder. Thus, the combination of — .50 D. sph. + 1.00 D. cyl., ax. 90 refracts rays as a cross cylinder; the — .50 D. sphere diverges rays at all meridians, and the superimposed + 1.00 D. cylinder, axis 90 converges all horizontal rays to an equal distance in front of the original point of focus. Thus, only the vertical rays continue to be dispersed by the — .50 D. sphere. In other words, the function of the minus sphere has been changed to that of a minus cylinder. One cannot employ a simple cylinder to test the accuracy of a sphere.

Book Reviews

The Art of Seeing. By Aldous Huxley. Price $2.50. Pp. 143. New York: Harper & Brothers, 1943.

(Reprinted from British Medical Journal)

Aldous Huxley had bilateral keratitis at the age of 16 which left him with greatly reduced vision. As is common in such cases, his sight improved towards middle life. After he had become an ardent devotee of the Bates method of visual training his vision improved so that it was "twice as good as before." To repay a debt of gratitude to his benefactor and to make his art more widely known, Huxley has ventured into medical literature and written a book—*The Art of Seeing.*

Bates's hypothesis is, of course, well known to the medical profession: visual derangements and refractive errors are due to a deformation of the eyeball by a condition of nervous and muscular strain in the extra-ocular muscles; for this reason, according to Bates, the refraction may change "a dozen times or more in a second," if a child tells a lie, and so on. According to Huxley "disappointed love" can produce refractive errors; and myopia in children, while it may be influenced by physical factors, is essentially due to the fact that school children "are often bored and sometimes frightened . . . so that they emerge from the educational ordeal with myopia or some other defect of vision." The universal treatment in all cases is the induction of cerebral and ocular "dynamic relaxation," on the achievement of which the eye becomes normal. This desirable condition is attained by exercises which include palming (covering the closed eyes with the palms of the hands and imagining a beautiful scene), butterfly blinks, relaxed breathing, sunning ("letting go and thinking looseness" and turning the eyes to the sun); swinging (rhythmic swaying of the body while looking out of a window to make the mind "friendly to movement" and "soothe" it "as do the movements of the cradle"), nose-writing (writing with an imaginary pencil attached to the end of the nose), flashing with dominoes, shifting, rubbing and kneading the upper part of the nape of the neck, and a host of other procedures which Huxley admits are undignified but which he assures us permanently help the vision.

Whatever be the value of the exercises, it is quite unintelligent of Huxley to have confused their advocacy with so many misstatements regarding known scientific facts. It has been shown that the hypothesis upon which these methods of treatment are based is wrong; but Huxley, while admitting he is ignorant of the matter and unqualified to speak, contends that this is of no importance because the method works in practice and gives good results; it comes into the category of "art," not of "science." This argument is perfectly allowable, for in other spheres than medicine empirical methods have often produced effective results the rationale of which may be mysterious. The most stupid feature about his book, however, is that he insists throughout on the physiological mechanism whereby these exercises are supposed to work. It would at least have been logical if he had continued to allow the reader to assume that he was speaking in ignorance of anything except results. He only borders on the ridiculous when he says that these methods result indirectly in the relief or cure of many serious diseases of the eyes, such as glaucoma, cataract, iritis, or detachment of the retina, because they "reduce nervous muscular tension, increase the circulation and bring back the *vis medicatrix naturae* to its normal potency." Like the bone-setter who treats by forcible manipulation every joint whether rheumatic or tuberculous, or most obsessionists who believe that every virtue resides in one particular drug or practice, he treats all visual troubles as one all-embracing unity, all amenable to one sovereign remedy.

There is, of course, another side to the argument. There would appear to be no doubt that these exercises have done Aldous Huxley himself a great deal of

good. Every ophthalmologist knows that they have made quite a number of people with a similar functional affliction happy. And every ophthalmologist equally knows that his consulting-room has long been haunted by people whom they have not helped at all. Huxley is quite right when he says that there is a tendency for the ordinary person, when there is a falling-off in vision, to "hurry off to the nearest spectacle shop" and there get fitted for a pair of glasses by some one who has "no knowledge of him as a physical organism or as a human individual." Nothing could be worse medicine than the indiscriminate and mechanical prescription of spectacles. He is quite right in saying that visual disabilities are often muscular and often psychopathic in origin. But every competent ophthalmologist treats the former with curative exercises (of a more scientific type than those recommended here), and, in the second case, at the least should advise the patient of the origin of his difficulties and suggest steps for their amelioration. For the simple neurotic who has abundance of time to play with, Huxley's antics of palming, shifting, flashing, and the rest are probably as good treatment as any other system of Yogi or Coué-ism. To these the book may be of value. It is hardly possible that it will impress any one endowed with common sense and a critical faculty. It may be dangerous in the hands of the impressionable who happen to suffer from glaucoma or detachment of the retina, and undoubtedly will be dangerous in the hands of the anxious parent of a myopic child who may be misled into neglecting the fundamental medical problems of growth, constitution, endocrinology, and rational ocular care, and may be persuaded to subject it to dangerous eye-strain in the absence of spectacles. But the greatest value of the book will be to the psychiatrist as an intimate and revealing self-study in psychology.

STEWART DUKE-ELDER.

Transactions of the American Ophthalmological Society. Seventy-Eighth Annual Meeting. Volume 40. Pp. 568. Philadelphia: American Ophthalmological Society, 1942.

These transactions present a stately volume of the twenty-three papers read at the annual meeting, Hot Springs, Va., June 1 to 3, 1942, under the presidency of the late Dr. Allen Greenwood. Among the subjects and authors may be mentioned: "Practical Importance of Aniseikonia," Dr. Edward Jackson; "A Reply to Criticisms of Aniseikonia," Dr. Walter B. Lancaster; "The Effect of Mydriatics on the Intraocular Pressure in So-Called Primary Wide Angle Glaucoma," Drs. Peter C. Kronfeld, H. Isabelle McGarry and Homer E. Smith; "Some Observations on the Results of Desensitization in Tuberculous Iritis," Drs. E. V. L. Brown, Ernest E. Irons and S. L. Rosenthal; "Experimental Studies in Ocular Tuberculosis," Drs. Alan C. Woods and Earl L. Burky; "Allergic Keratoconjunctivitis," Drs. C. S. O'Brien and J. H. Allen; "Corneal Vascularization Problems," Drs. Derrick Vail and K. W. Ascher; "Local Sulfonamide Therapy Catarrhal Conjunctivitis," Drs. Phillips Thygeson and Alson E. Braley; "Precancerous Melanosis and Malignant Melanoma of the Conjunctiva and Skin of the Lids," Dr. Algernon B. Reese; "A Colored Reflex from the Anterior Capsule of the Lens Which Occurs in Mercurialism," Dr. Walter S. Atkinson; "Fibroblastic Overgrowth of Persistent Tunica Vasculosa Lentis in Infants Born Prematurely," Dr. T. L. Terry, and "Studies of the Fields of Vision in Cases of Rathke Pouch Tumors," Drs. Henry P. Wagener and J. Grafton Love. Some of these articles were also published in the ARCHIVES.

The volume concludes with the theses of the newly elected members: "Progressive Myopia: A Possible Etiologic Factor," Dr. Edwin W. Burton; "Endophthalmitis with Secondary Glaucoma Accompanying Absorption of the Crystalline

Lens," Dr. Robert H. Courtney; "Further Observations on Autofundoscopy (Auto-Ophthalmoscopy of Eber; Purkinje Figure of Walker)," Dr. Leslie C. Drews; "Herpes Zoster Ophthalmicus: Report of Cases and Review of the Literature," Dr. Ambrose Earl Edgerton; "The Ocular Pathology of Methyl Alcohol Poisoning," Dr. Walter H. Fink; "Transscleral Lacrimal Canaliculus Transplants," Dr. Glen Gregory Gibson; "An Experimental Study of Iridodialysis," Dr. George L. Kilgore; "The Use of Conjunctival Flap in Cataract Extraction," Dr. Walter Moehle, and "Objective Strabismometry in Young Children," Dr. Maynard C. Wheeler.

The new editor of the transactions, Dr. Wilfred E. Fry, is to be complimented on the excellent appearance of this volume.

ARNOLD KNAPP.